NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY

SUBMARINE BASE, GROTON, CONN.

REPORT NUMBER 985

SHAD-NISAT:

A COMPOSITE STUDY OF
SHALLOW SATURATION DIVING

incorporating

Long Duration Air Saturation with Excursions, Deep Nitrox Saturation,
and
Switch from Nitrogen to Helium

Edited by

R. W. Hamilton, G. M. Adams, C. A. Harvey
and
D. R. Knight

Released by:

William C. Milroy, CAPT, MC, USN
Commanding Officer
Naval Submarine Medical Research Laboratory
August 1982

1982 August

SHAD-NISAT:

A COMPOSITE STUDY OF SIMULATED SHALLOW SATURATION DIVING

incorporating

long duration air saturation with depth excursions, deep nitrox saturation, and an isobaric switch from nitrogen to helium

Edited by

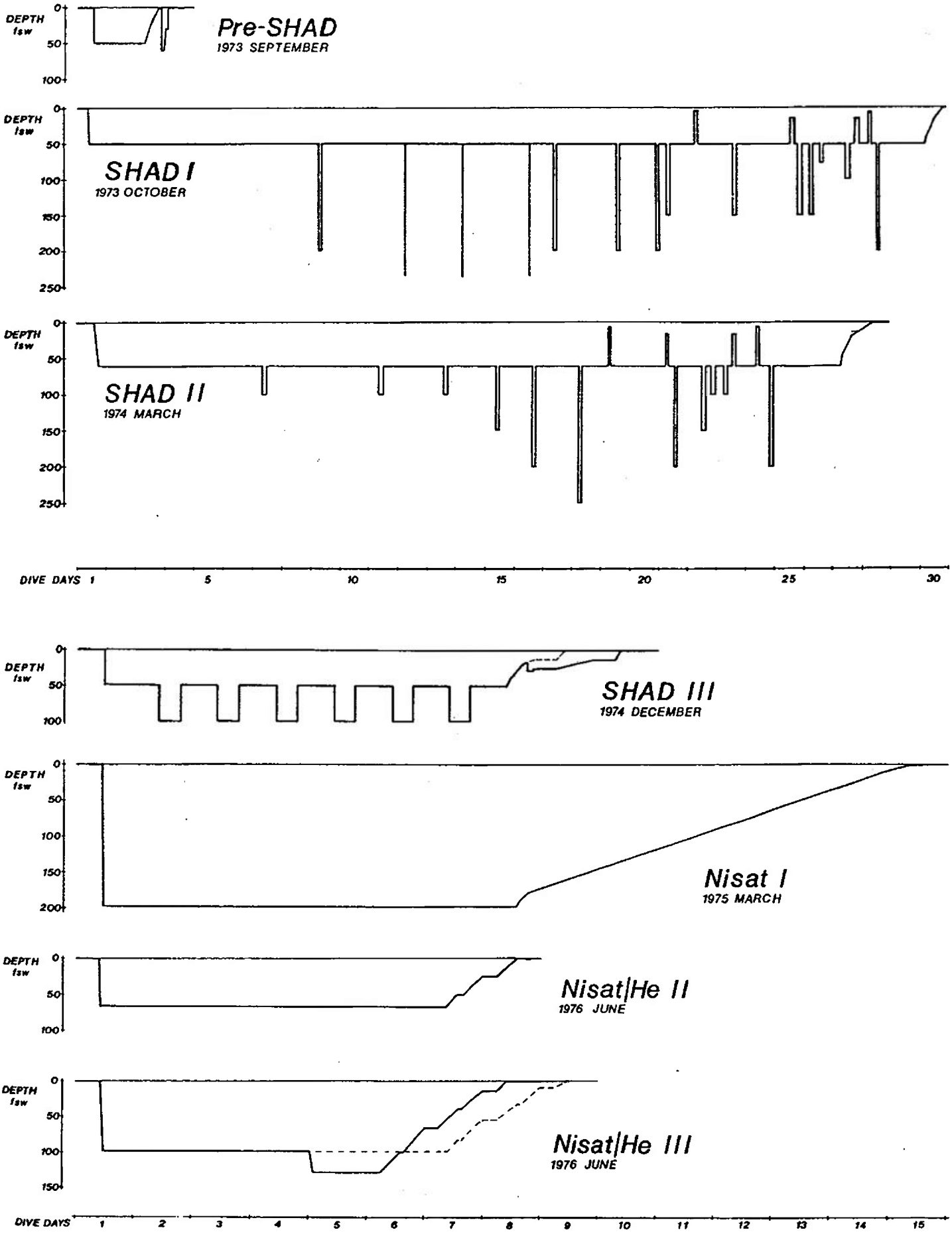
R.W. Hamilton, G.M. Adams, C.A. Harvey and D.R. Knight

Naval Medical Research and Development Command

Work units M4306.02-3114 BEK9.02
M4306.01-8013
MPN10.003-7060
MR041.01-5057
MF51.524.014-9018
M4306.01-2001BFM9
M4306.02-2110BAK9

Approved and released by

W.C. Milroy, CAPT, MC, USN
Commanding Officer
Naval Submarine Medical Research Laboratory



FRONTISPIECE: Profiles of the SHAD and Nisat dives. The plots are pressures in feet of sea water (scales at left) and dive days; the center scale applies to the upper three dives, the bottom one to the lower 4.

PREFACE

The American Shad (Alosa sapidissima) is a type of herring that inhabits the East and West Coasts of the U.S., both in fresh water during spawning, and according to Carleton Ray and Elgin Ciampi, in "mysterious sea locations" during the non-breeding season (Ray and Ciampi, 1956). Project SHAD was envisioned to remove some of the mystery, not of the American Shad, but of the acronym with the same spelling. The report deals with a complex series of diving and hyperbaric activities that have provided considerable insight into the mysteries of Shallow Habitat Air Diving (SHAD), which include excursion diving, human tolerance to high oxygen and exposure to hyperbaric nitrogen, and the results of the switch from one background inert gas to another when saturated. The name is particularly appropriate because shad frequent the Thames River on which the Naval Submarine Medical Research Laboratory is located, and are a favorite delicacy of the local population. Caught in gill nets, usually at night, the 4-pound fish have tasty fillets which can be removed from the plentiful bones by a deft technique, and the females also have a delicate roe.

SUMMARY PAGE

PROBLEM

Naval personnel, while conducting military missions in the undersea environment, may intentionally or accidently become confined within an environment of hyperbaric air for hours to weeks. Heretofore, the tolerance for such environments could not be predicted from Naval operational experience nor civilian occupational exposures. Further; there was limited information on how to work with and use these environments.

FINDINGS

The NSMRL conducted a series of hyperbaric chamber research experiments in which Naval volunteers lived either in compressed air or nitrogen-oxygen mixtures beyond the time that their body tissues became saturated with the gases. It was found that humans can safely live in hyperbaric air at pressures equivalent to 50 and 60 feet of depth for up to 30 days without incapacitation by pulmonary oxygen toxicity, and useful excursions to greater and shallower depths can be made, but some oxygen toxicity can be expected under certain circumstances. Life was not as comfortable at a pressure of 7 atmospheres in nitrogen with a normal pressure of oxygen; subjects developed nausea and vomiting a few hours after beginning the exposure, and these symptoms were accompanied by decrements in task performance. Slight elevation of the oxygen level to 0.3 atm was associated with improvement of the distressful symptoms. After recovery from the nausea, it was found that the performance in this very dense atmosphere of exercise was poorly tolerated by the subjects in comparison to their exercise performance at sea level. Near-normoxic nitrogen was not at all unpleasant at shallower depths of 66 and 99 fsw, where the subjects felt mildly lethargic. Such lethargy cleared when the subjects made a sudden transfer from the nitrogen environment to a helium environment at the same storage depth and oxygen level. Symptoms of itching, skin rashes and joint pains occurring within hours of the isobaric shift to heliox resembled decompression sickness, and were relieved by recompression therapy.

APPLICATION

The NSMRL chamber experiments provided the Navy with validation of tables for excursions, and for decompression from saturation exposures with air and "nitrox." Confirmation was made of predictions that isobaric shifts from nitrox to heliox can produce symptoms of decompression sickness in relatively mild hyperbaric exposures. Equally important was the accumulation and publication of valuable scientific information concerning the human tolerance for multiday exposures to hyperbaric nitrogen and oxygen.

ADMINISTRATIVE INFORMATION

This report covers a series of projects carried out at NSMRL during the period 1973 to 1976, principally, under work units M4306.02-3114 BEK9.02, M4306.01-8013, MPN10.003-7060, MR041.01-5057, MF51.524.014-9018, M4306.01-2001BFM9, M4306.02-2110BAK9. The report was prepared by Dr. R.W. Hamilton working as a consultant to the American Institute of Biological Sciences under contract N00014-75-C-0348.

ABSTRACT

Hamilton, R.W., G.M. Adams, C.A. Harvey and D.R. Knight, editors. SHAD-Nisat: A composite study of shallow saturation diving. NSMRL Rept. 10XX. Groton, CT: Naval Submarine Medical Research Lab., 1982.

Seven dry chamber laboratory exposures at NSMRL explored the feasibility, physiological effects, safe limits, and operational procedures applicable to air and nitrogen-oxygen saturation diving. Objectives included establishing Navy capability in nitrogen-based saturation diving and improving certain submarine rescue procedures. The four SHAD dives looked at air for breathing in saturation at 50 and 60 fsw. Excursions from these depths demonstrated possible work procedures, covering depths ranging between 5 and 250 fsw and for times as long as 8 hours. The exposures were well tolerated but disclosed problems with oxygen toxicity in daily 8-hour excursions to 100 fsw and an increased sensitivity to extra oxygen breathing in several of the divers. The long air exposures caused red blood cell losses; recovery began a few days after return to normal pressure. The divers were also deconditioned, presumably because of the confinement and several weeks of inactivity. Decompressions from descending excursions were free of bends but some ascending excursions caused itching and ultrasonically-detectable bubbles. Two of three divers became nauseated 3 hours after beginning a saturation exposure in an atmosphere containing 0.22 atm oxygen, balance nitrogen, at 7 atm abs; the sick divers felt better after PO_2 was raised to 0.3 atm. All 3 of these divers were "drunk" for several days as a result of nitrogen narcosis, but recovered many aspects of normal performance after 5-6 days. In two experiments divers saturated with nitrox (0.3 atm PO_2) at 66 and 99 fsw were switched to a helium mixture. Itching followed within 3 hours, and was quite intense for the 99 fsw crew; one of these had to be treated by recompression for "counterdiffusion sickness" manifested as knee pain. Hyperbaric bradycardia was observed in most of the divers, intensifying on the deeper excursions.

* ADAPTATION / BRADYCARDIA / COUNTERDIFFUSION / BREATHING MIXTURES / DECOMPRESSION / DECOMPRESSION SICKNESS / ECG / EEG / ERYTHROCYTES / EXCURSION DIVING / HELIUM / HYPEROXIA / ITCHING / NARCOSIS / NAUSEA / NITROGEN / OXYGEN TOXICITY / PERFORMANCE / SATURATION / SELECTION / TABLES /

ACKNOWLEDGEMENT

There was hardly a person in the Naval Submarine Medical Research Laboratory who was not touched in some way by these projects, and many others elsewhere on the Submarine Base and in other Navy locations made significant contributions. It is impossible to recognize them all by name. We thank in particular the divers and investigators who served as subjects, the chamber crew who performed the operations, the medical officers who stood watch, the laboratory personnel who helped produce the data, and the many independent support personnel. We particularly recognize the foresight of CAPT E. Fisher Coil and the Bureau of Medicine and Surgery whose strong support--in many ways--made it all possible. Drs. Al Behnke, Chris Lambertsen and Chuck Shilling served as advisors.

The name SHAD was conceived by CDR Ray Sphar, whose leadership as Commanding Officer of NSMRL was critical to the inception and successful completion of the project. Our thanks to CAPT Bob Margulies who helped make this report possible.

CDR Bill Hunter deserves credit for holding the SHAD-Nisat data together as he carried on with the successor dive series, Airsat; special thanks go to him and to CAPT Bob Margulies for conceiving and implementing the idea of doing this report.

While some of the information given here reflects published material, much has been drawn from notes, records, data and unpublished reports of individual investigators, as well as numerous meetings and interviews. We have defined authorship of a few of the sections, but we are particularly grateful to those folks who have helped with all the others. For background and policy in Chapter I, Bud Carey; for the facility descriptions and operations, Jim Jordan; for diver profiles, Bill Mooney; for the medical plans, Dr. Roger Williamson and several others; for human factors, George Moeller; for physiology, the late Karl Schaefer and Jim Dougherty; for vision, Jo Ann Kinney, Chris (McKay) Schlichting and colleagues; for biochemistry, Don Tappan, Elly Heyder and Mike Jacey; for psychology, Ben Weybrew; for the detailed "debriefing" report of SHAD III, Dave Miller; for help with decompression computations, Ruport Hester and George Moeller; for organizational management throughout the projects, Jim Parker; for locating old photographs, Wally Abrams (and thanks to the original photographers); for proofreading and managing production of this report, Terry Smith; and to many others for help in many ways. The decompression computations obtained from Union Carbide Corporation were prepared by Dave Kenyon with the assistance of Mark Freitag. Brian D'Aoust of Virginia Mason Research Center performed animal experiments which proved critical to the safe execution of the Nisat/He experiments.

This report was prepared by Dr. R.W. Hamilton, Hamilton Research Ltd., Tarrytown, NY.

TABLE OF CONTENTS

LIST OF TABLES AND FIGURES

I.
INTRODUCTION

The U.S. Navy relies on helium for conducting its saturation diving, but helium is expensive and involves complicated logistics. The use of air or nitrogen-oxygen mixtures for saturation diving offered an attractive alternative, but at the time of these SHAD-Nisat experiments little was known about either the techniques, the hazards, or the physiological limits of this approach. Commercial diving had made little use of air-nitrox saturation methods, partly because of lack of knowledge of such things as the role of oxygen toxicity, nitrogen narcosis, decompression, and possible limits to exercise when breathing the dense gas. Operational knowledge and understanding on these topics was needed for both the Navy's diving and submarine rescue missions, and this was the incentive for NSMRL to undertake this series of pioneering tasks.

This report covers seven hyperbaric exposures on several themes; these include long duration saturation in air with both short and long excursions, saturation with a nitrogen-oxygen mixture at an extreme depth, and a switch of gas under saturation from nitrogen to helium as the inert background gas. In addition to experience with excursions, saturation, and gas switching, the projects produced significant data on long duration exposures to high oxygen levels and adaptation or accomodation to the effects of nitrogen narcosis.

One might wonder why so many objectives are covered and why the project has two names. SHAD and Nisat were in fact different projects with different principal investigators; the history of the projects is given in section I.C below. While there was an early interest in undersea habitats, the unifying theme for all of these experiments is submarine rescue. All objectives and most of the findings of the SHAD-Nisat experimental series deal with the specific problems which may be encountered in the present submarine rescue scheme or needed in the development of future or alternative plans.

A multi-laboratory approach was taken. The Virginia Mason Research Center, Union Carbide Corporation's Environmental Physiology Laboratory, the Institute for Environmental Medicine at the University of Pennsylvania and several U.S. Navy Diving Commands (including Submarine Development Group One and the Naval Undersea Systems Center) cooperated with NSMRL in support of the program, offering advice, computations, animal work, personnel support and equipment.

This is an after-the-fact report, written some 5 years after the last of the experiments. It uses the common aspects of the various themes to tie together the loose ends of these experiments into a single report covering the overall objectives and summarizing the findings. Many elements of the program have been reported by investigators with various disciplines; these are recognized and referenced here. The report includes details of the dive series, some as-yet-unreported data, and general conclusions. We have attempted to put the results of the program into perspective in light of the present state of the art.

A. Background

1. The habitat diving concept

In the diving sense "saturation" is the state where a diver's body is equilibrated with the ambient gas. A more practical definition is the state in which the diver's decompression time for that depth (and that inert gas exposure) is at a maximum and will not increase with additional time at depth. Saturation permits divers to live at or near the depth of their work. They may live on deck in a diving chamber and transfer to and from work in a pressure capsule in a "habitat" on the sea floor.

The chamber or habitat may be at any depth within the safe diving range as yet explored. This report and the projects it covers are concerned with relatively shallow diving, in the air range, where limits are not as well defined as they are with deeper heliox saturations. The practice of working out of a chamber on the bottom of the sea is referred to as "habitat diving." The practice gives a dive team continuous access to a work site. They may work at the same level or by use of special procedures may excurse up or down, or out away from the habitat.

While the habitat diving concept increases capability, it is not without its hazards. In addition to the obvious operational problems of living in the sea (i.e., drowning, cold, navigation, etc.) the divers must live in an artificial gaseous atmosphere and tolerate pressure variations. The SHAD projects considered that some baseline data on habitat diving was available; they sought details that would first permit an extension of the concept, but would also serve to bring this technology into the Navy's armamentarium. One distinct new approach was the use of compressed air in a range beyond established experience. The use of air offered economics and logistic advantages, but entailed new levels of the familiar problems of oxygen toxicity, nitrogen narcosis and decompression.

For Nisat the habitat concept was expanded into another problem area, with related physiology but quite a different operational objective -- the rescue of a submarine crew exposed to increased air pressure. This also touched on an area of practical interest in habitat diving, having a diver saturated with air (or a nitrogen mixture) excurse using a helium-oxygen mix. Thus two objectives were served in Nisat, excursions from a habitat to depths well beyond the air range, but more importantly knowledge about an inert gas shift during submarine rescue.

2. Situation at the time of the experiments.

The first serious at-sea saturation dive was conducted by Dr. Edwin A. Link in 1963. After an 8-hour exposure at 60 fsw with himself as a subject, Link put a single diver 200 feet deep in the Mediterranean for an overnight saturation in a cylindrical, sealable, submersible decompression chamber, and decompressed him on deck in a deck decompression chamber or DDC (Link, 1963). This was followed soon after by the first of Cousteau's saturation habitat operations (Cousteau, 1964; Chouteau, 1969) and the U.S. Navy's Sealab program (MacInnis and Bond, 1969).

Although the benefits of saturation diving were appreciated by Haldane and probably even by Paul Bert, the first practical advocate seems to have been Behnke (1942), who proposed saturation as a means of solving some of the decompression problems of tunnel workers. End and Noble spent 27 hr in air at 101 fsw in 1938 in a hospital recompression chamber in Milwaukee (Kindwall, 1975). It remained, however, for Bond and his group at the Naval Submarine Medical Research Laboratory (NSMRL) through the Genesis experiments (Bond, 1964; Bond, 1966; reviewed by Koblick and Miller, in press) to make the idea a reality. The Genesis experiments showed that the lethal aspect of attempting to live saturated in hyperbaric air were clearly due to the toxicity of the oxygen in the air, and they followed through to show it could be done with both animals and man when the oxygen toxicity was controlled (Workman, Bond and Mazzone, 1962). After "waiting a discreet period" (which he admits was "about 45 minutes") before jumping onto Bond's idea, Cousteau proceeded with his Conshelf series. Overlapping with Conshelf, the Sealab series led by principal investigator "Papa Topside" Bond clearly proved the practical aspects of saturation diving. Their work was all oriented toward sea floor habitats, but commercial saturation diving has taken the "commuter" concept; nevertheless, it was Bond, Cousteau and Link who made it all happen.

The Sealab, Conshelf, and Man-in-Sea projects had clearly shown the feasibility of sea floor habitat diving, but some of the limitations had come to the surface as well. Sealab in particular operated with a most unrealistic restriction on the permissible excursion distance which the divers were allowed. The Sealab divers were limited to a 25 fsw* vertical excursion distance from the saturation storage depth. It was suspected at the time that larger distances could be used safely; one clue that this was tolerable was the successful use of an initial "pull" at the beginning of a saturation decompression. In the Conshelf series an excursion to the deeper station (see Chouteau, 1969) was made but these excursions were quite conservative -- they used essentially surface diving limitations.

The first experiment to show clearly that an excursion from saturation could be for a greater distance than from the surface was the work of Larson and Mazzone (1967). This experiment, though a relatively short and simple one, was another significant NSMRL step in the development of habitat diving technology.

Larson and Mazzone's work took a "Boyle's Law" approach. Another way of stating this is as Haldane ratios; if the base depth or denominator of the Haldane ratio increases, the allowable dive for given decompression will also increase. Boyle's Law states that the volume of a parcel of gas (a bubble, say) is inversely proportional to its pressure, and that a change in such a volume is a function of the relative change in pressure. Thus a given excursion would have less effect on bubble size if the dive started from some water depth rather than from the surface. Boyle's Law is not the whole story, but it now seems well established that deeper and longer excursions can be made safely from an undersea habitat than from the surface.

---

* The primary unit of pressure used in this report is the foot of sea water, fsw, defined as 1/33 of a Standard Atmosphere.

The Tektite experiments (Miller and Lambertsen, 1971; Miller, et al, 1971) demonstrated another type of habitat diving, this time in the shallow range and using a nitrogen-oxygen or "nitrox" mixture instead of air. The related Predictive Studies II at the University of Pennsylvania (Lambertsen and Wright, 1973) showed under a rather comprehensive biomedical scrutiny that saturation at a depth of 100 feet of pressure in a nitrox atmosphere was tolerable. Experiments by Edel were intended to show that emergency surfacing was a feasible alternative if needed from the shallow habitat (Edel, 1971).

NSMRL participated in providing background material for Tektite by a series of nitrox saturation dives conducted in the Genesis chamber. In 5 saturations with 13 subjects at a depth of 40 fsw, normoxic, it was shown that the "classical" values used for computing decompressions with nitrogen were grossly inadequate, and that more conservative values would have to be used (Markham, 1970).

The last precursor to SHAD and a major impetus to it was the NOAA OPS program (Hamilton, et al, 1973) in which NSMRL scientists participated. The NOAA OPS investigators gathered together the available information bearing on excursion diving and relevant decompression, developed a plan for computing excursions, and carried out a series of air excursions from nitrox saturation. The Union Carbide-Ocean Systems team which performed NOAA OPS was primarily operationally oriented, but NSMRL provided extensive biomedical monitoring throughout the series. This coverage included human factors, blood chemistry and hematology, visual evoked response, Doppler monitoring, bone necrosis x-rays, gas exchange, and pulmonary function, and the divers were all given a thorough initial physical exam through the NSMRL Longitudinal Health Study (Sawyer and Baker, 1972; Tansey, 1974).

The involvement of NSMRL in NOAA OPS was significant for both projects, NOAA OPS and SHAD-Nisat. The biomedical coverage of the NOAA OPS divers provided by NSMRL strengthened the conclusions about the suitability of the concept, but also the idea for SHAD was enhanced by that involvement. The habitat diving concept so enthralled NSMRL investigators working in Tarrytown on NOAA OPS that in the face of a cutoff of travel funds the NSMRL investigators completed their experimental programs, paying their own travel expenses.

One significant new element to SHAD that distinguished it from NOAA OPS, Tektite, Predictive Studies II and the other dives that had been done before should be mentioned. These dives all used nitrogen-oxygen or nitrox mixtures in the habitat as the saturation storage gas. SHAD explored the use of air as the storage gas. This had the possibilities of substantially broadening the application and reducing the operational complexity of this diving mode.

Other details about the technical background of habitat diving at the time of SHAD is well covered in the reports of the NOAA OPS project (Hamilton et al, 1973; Miller et al, 1976*).

B. History of the SHAD and Nisat projects

The concept for shallow habitat diving had been around for some time. It was now time for this to be made relevant to Navy needs. Planning for the first SHAD dive began in the latter part of 1972. The project originally carried the tentative name Aura, which in Latin means "air in motion" or "to breathe." From the beginning a long dive was planned, with air as the saturation gas and with excursions to be made on air as well. (The relevance of the name SHAD is discussed in the preface.)

Some significant barriers had to be overcome. For one, human experimental work in diving had not been done for some time at NSMRL, and during this period a new national ethic on protection of human subjects had emerged. It was necessary to secure clearance from the Secretary of the Navy before the exposure could be carried out or even funded. This SECNAV approval was received on 21 Feb 1973.

Further, there was still serious doubt in the minds of some medical people that the concept was safe. Animal experiments had to be carried out to show that it was. These were accomplished, first with an exposure of rats for 60 days at 50 fsw, then a 36-day exposure at 60 fsw (Murray, et al, 1974; Jacey and Tappan, 1974; Ferris and Shaffer, 1974; Heavers et al, 1973). Results of these tests were convincing -- there was no pathology or any defects that looked at all likely to cause any damage to humans, and even very little evidence of adaptative changes. There was a predicted red blood cell loss due to oxygen, and some other evidence suggesting stress.

With the high sensitivity of this operation the NSMRL command felt that especially thorough preparations needed to be made to deal with virtually any emergencies that could arise during the dive, and that this plan should be well thought out and documented. This was done; the essence of this comprehensive plan is given in the section on medical preparations.

Contracts were made with the laboratory at Union Carbide Corporation, Tarrytown, NY, to compute the excursion profile for the first 30-day experiment and to provide decompression procedures for use in emergencies and in case there were deviations from the profile. A saturation decompression table was also computed on specifications agreed upon by both groups.

Engineering and mechanical work on the chambers was carried out during 1973 to bring the support equipment to readiness for long human experiments. Things appeared in order, but a "habitability" dive was carried out to "shake down" the facility and the operation, and to verify the saturation decompression profile. This dive, Pre-SHAD, gave the SHAD principal investigators an opportunity to evaluate their experimental medium, disclosed some difficulties in running the chamber (humidity was high), and showed that the planned SHAD decompression was inadequate by causing bends in both divers.

This was followed (after some corrections) by SHAD I, in October of 1973. SHAD I consisted of a long air saturation at 50 fsw with some 18 excursions scattered over that period. Excursions were both "up" and "down" from the habitat depth, from near surface to 235 fsw in depth; they were for durations of up to 60 minutes and were all without stops.

A listing of all the dives covered in this report is given in Table I-1. The "experimental period" spans the first to the last data point, but many parameters were monitored for shorter times.

SHAD I went well and proved the concept of saturation on air at 50 fsw. This confirmed the plans to carry on with SHAD II and extend the saturation depth to 60 fsw. This second SHAD dive was begun in March of 1974. The pattern for SHAD II was similar to SHAD I, with a habitat level of 60 fsw (air) and 15 excursions covering the range of 5 to 250 fsw over 26 days. The same monitoring pattern was followed. Operationally SHAD II was uneventful but the insidious onset of what might loosely be called "hematological oxygen toxicity" was a matter of concern. There was now reason to doubt that the logical next step to 70 fsw could be done without real problems.

SHAD I and II were conceived and the project led by Medical Service Corps officer LCDR George M. Adams, who had the official function of "Project Coordinator" but by all recognized criteria should be considered the Principal Investigator.

The next step was uncertain. This and other factors, among which was that Mr. Adams was not a Medical Officer, led to project responsibility being handed over to Dr. Claude A. Harvey, LCDR, MC, USN. He became coordinator and medical supervisor of SHAD III and was Principal Investigator of the Nisat series.

SHAD III, held in December 1974, took a more pragmatic approach to the excursion, simulating a full work day. Eight-hour excursions to 100 fsw from a base of 50 fsw were conducted every day for 6 days, all with air. The decompression profile for SHAD III (and the Nisat dives) was calculated by Dr. Harvey with the assistance of the Naval Undersea Systems Center across the river in New London.

The first Nisat dive was designed to verify that saturation for 7 days at 7 atmospheres and its ensuing decompression could be carried out in case treatment at this depth was needed in subsequent Nisat dives. It was run in March of 1975.

The last two Nisat dives carry the designation Nisat/He in this report, to show that a switch to helium was used. These two dives were designed to emulate possible rescue profiles of the DSRV (Deep Submergence Rescue Vehicle) in case a crew of rescued submariners happens to be saturated with air. Depths of 66 and 99 fsw (3 and 4 atm abs) were used. These two dives were both done in June of 1976.

Adams had left the Navy earlier, and within a month following the third Nisat dives Harvey departed NSMRL. Most of the work of individual investigators has been reported, but nothing in the way of an overall project report has been issued prior to this one. The foregoing tells the general story of SHAD-Nisat; more details on specific objectives and their rationale follow.

Table I-1
Summary Chart: SHAD and Nisat Experiments

Dive; Year	Exptl period; t at pressure	Bottom time	Decomp time	Divers	Description; saturation depth, gas; excursions; DCS; etc.
Pre-SHAD 1973	10Sep-14Sep	2 days	10 hr	1. GA 2. TT	Habitability test, no investigations; 50 fsw, air; DCS in both divers, at surface.
SHAD I 1973	17Sep-30Nov 1 Oct-30Oct	29.5 days	13.5 hr	1. WB 2. SW *	30-day saturation w/excursions; 50 fsw residence depth, air; 18 air excursions to depths from 5 to 235 fsw.
SHAD II 1974	25Feb-3May 15Mar-12Apr	26 days	28 hr	1. GS 2. RF **	26-day saturation w/excursions; 60 fsw residence depth, air; 15 air excursions to depths from 5 to 250 fsw.
SHAD III 1974	18Nov-30Dec 3Dec-12Dec	7 days	46 hr	1. DM 2. PP 3. RO	Saturation w/long excursions; 50 fsw residence depth, air; 6 daily 8-hr air excursions to 100 fsw. DCS during sat'n decompression at 18 fsw.
Nisat I 1975	4Mar-2Apr 12Mar-25Mar	7 days	7 days	1. NT 2. JB 3. RJ	Deep nistox saturation; 198 fsw; normoxic nitrogen-oxygen mix; no excursions.
Nisat/He II 1976	31May-22Jun 8Jun-15Jun	6 days	28.7 hr	1. JC 2. MH 3. RO	Inert gas switch; 66 fsw saturation, nitrox; isobaric gas switch from normoxic nitrogen to helium on 4th dive day.
Nisat/He III 1976	17Jun-7Jul 23Jun-30Jun	5 days	54.3 hr	1. TG 2. RE 3. RL	Inert gas switch; 99 fsw saturation, nitrox; isobaric gas switch from normoxic nitrogen to helium on 4th dive day; DCS after switch.

* SHAD I control subjects
c1. PD (thru Oct 2)
c2. PP (Oct 10 on)
c3. GS

** SHAD II control subjects
c1. WB
c2. SW (Mar 7 on)
c3. TW (thru Mar 15)

C. Project objectives

The SHAD-Nisat experiments covered in this report constitute a series of more or less distinct elements converging on a few broad objectives.

1. Overall objectives

The SHAD experiments were designed to assess the biomedical feasibility of man's extended residence in compressed air at relatively shallow depths and his ability to perform in excursions above and below the fixed residence depth.

In contrast to this general goal, Nisat had a crisply pragmatic bent, to assess survival capability of submariners trapped in a compressed air environment who might be required to switch to a helium-oxygen atmosphere during rescue.

2. Specific objectives

Each of the dives fit into this pattern in a specific way, with the environment that was simulated and the work that was done experimentally.

a. Pre-SHAD: Habitability

Pre-SHAD was conducted to confirm the "habitabilty" of the Genesis chamber and to check the saturation decompression schedule proposed for use in the longer SHAD runs. Habitability here comprised more than just the bunk, shower and environmental control, but included also the procedures for managing shifts and maintaining the chamber system around the clock. A significant but unstated purpose was to give the Principal Investigator a chance to "be the first" to tolerate the exposure.

b. SHAD I and II: Long air saturation with excursions

These exposures fit the overall SHAD objective of biomedical and performance assessment of habitation in compressed air at shallow depths with upward and downward excursions. The exposures were relatively long by ordinary diving standards, about one month. SHAD I tested habitation at 50 fsw, and SHAD II was set at 60 fsw. Both involved excursions in both directions--deeper, and back toward the surface--covering a variety of excursion distances and times within the no-decompression (i.e., no-stop or NOAA OPS) limits. The oxygen effects of the 60 fsw exposure were also of interest.

c. SHAD III: Air saturation with long excursions

The pattern changed slightly for SHAD III. This dive like SHAD I was set at 50 fsw, but the excursion pattern was to simulate a working day at the excursion depth of 100 fsw. The excursion was repeated daily for 6 days. To evaluate the excursion decompression and the divers' response to this level of hyperoxia were secondary objectives.

d. Nisat I: Deep nitrogen-oxygen saturation

The first Nisat dive was to verify that decompression could be carried out from the deepest depth that might reasonably be needed for treatment following a nitrox-heliox switch. A secondary objective was to study the effects of several days exposure to high nitrogen pressures.

e. Nisat/He II and III: Nitrogen to helium switch

These dives were to investigate the effects of switching the background inert gas in saturated divers from nitrogen to helium, at progressively increasing depths. These two were at 3 and 4 atm abs., 66 and 99 fsw. The motivation for this switch was its possible need in submarine rescue.

D. The specific exposures and their rationale

Whatever the objective, the series provided exposure to several environmental stressors. The parameters chosen were a compromise of various factors; the factors and the logic we followed in selecting them are given in the following sections.

1. Oxygen toxicity, obvious and insidious

The long exposures to compressed air in SHAD provided a test of the existing criteria for long term pulmonary oxygen toxicity. Central nervous system (CNS) toxicity was not a factor in any exposures except perhaps in the deep excursions (250 fsw, PO_2=1.8 atm); for the duration and level of exercise used in these excursions no CNS problems were expected (Clark and Lambertsen, 1971) and none was seen.

The level of oxygen partial pressure which had been generally accepted as safe for continuous exposure was 0.5 atm (Clark and Lambertsen, 1971). This level was chosen for SHAD I. SHAD II used a slightly higher level (PO_2=0.58 atm), thereby extending this limit. It has been determined in space experiments that exposures to slightly higher oxygen levels for similar durations in the absence of inert gases were safe, and there was no reason to believe that the compressed air environment would be any more stressful. The animal exposures carried out in preparation of SHAD (Murray et al, 1974; Jacey and Tappan, 1974) showed no pathology or abnormalities at 50 and 60 fsw, for periods longer than was planned for SHAD.

The SHAD III profile of daily 8-hour excursions to 100 fsw from 50 fsw saturation was chosen to provide a meaningful work capability using excursion techniques. It resulted in an oxygen exposure that exceeded the limits defined by the CPTD approach to management of lung oxygen toxicity as defined by Wright (1972; CPTD is discussed in more detail in Chapter VII).

We justified this exposure in several ways. First, the CPTD exposure "limits" were levels that might cause a measurable decrease in vital

capacity, but one that we felt would be easily tolerated. The development of pulmonary toxicity is gradual, and in the event that a given subject proved to be unusually sensitive to oxygen it would still be possible to stop the progress of the toxicity effects at any time by reducing the oxygen level. The continuous daily monitoring of biochemical as well as pulmonary parameters was expected to reveal any disturbances as they might develop. The success of SHAD I and II--exposures, monitoring, and operations--added to our confidence that the risk was acceptable in SHAD III. The Nisat experiments did not involve hyperoxic exposures.

2. Effective excursions

SHAD I and II were planned to simulate a diversity of excursions which might be used on habitat operations. The excursions were scattered about the day and evening to observe any diurnal effects on performance; their frequency was intensified toward the ends of the saturation periods. They were planned so as to minimize the effect of descending excursions on subsequent ascents. The excursions were planned by depth and time of occurrence, and durations were calculated according to the decompression criteria (see Chapter VI).

Excursions for SHAD III were planned to simulate a full work day of 8 hours and the desired excursion distance was calculated to be acceptable.

3. Nitrogen narcosis and adaptation to it

Traditional diving provided an experience base for the narcotic levels used in SHAD, and these had be quantitatively investigated in NOAA OPS. Narcosis during excursions was a subject of study but not a risk factor.

The 7 atm exposure of the first Nisat dive was unprecedented in its degree and duration. The objective was to demonstrate saturation and decompression capability in the event a deep treatment was needed following an inert gas switch planned for the later Nisat dives. We chose to go as deep as we could, as long as we could be sure the exposure would be well tolerated. One exposure to this depth and one slightly deeper had been made by DFVLR, the German Institute for Aviation and Space Research (Hartmann and Fust, 1966; 1967). The published reports have few details, but undocumented discussions revealed that they had had no problems at 7 atm but the divers had been "sick" at 8 atm. We chose 7 atm (198 fsw), feeling we could "retreat" back to 165 fsw in a short time if necessary.

Another aspect of the exposure to narcosis to be researched in the SHAD experiments and Nisat I was the question of "adaptation" to narcosis. There had been evidence that an individual continuously exposed to narcotic gas begins to recover from the performance decrements caused by narcosis within a few hours and may return to normal in a day or so (Schmidt et al, 1974). This is perhaps the same phenomenon as that affecting divers who make repeated daily dives and in the process become more tolerant of narcosis. It was planned that Nisat I would be long enough to allow physiological and psychological adjustments to take place, or until medical evaluation indicated further exposure would be detrimental to the divers.

4. Switch of inert gas

Nisat/He II and III were planned to evaluate the effects of counterdiffusion following an isobaric switch of inert background gas from nitrogen to helium. These were planned as "normoxic" to avoid confusion of the results by oxygen toxicity.

Originally plans had been to conduct the first switch at 99 fsw and the second at 132. The 132 fsw or 4 atm abs. level had been chosen arbitrarily by the Navy as the maximum level of exposure to compressed air which a submarine crew could presumably tolerate for the two-day period required to effect a rescue. The 99 fsw level was to be a step toward the deep one. Only a few days before going to pressure (at 99 fsw) in Nisat/He II we decided to make the first run at 66 fsw instead; this decision turned out to be appropriate. It was based on findings with goats at Virginia Mason Research Center that a significant number of Doppler detectable bubbles were found in goats following a similar switch (D'Aoust, 1977a).

E. Published material on SHAD and Nisat

From the time of the experiments up until the time of this report a number of publications have been issued that deal with the SHAD and Nisat experiments. Many of these reports deal adequately with parts of the experimental results and in a sense should be considered as "chapters" in this report. Results of the published material are summarized here, but in most cases we have not chosen to reproduce the data. For this reason the level of coverage of this report varies somewhat from topic to topic.

A listing of all the documents which are known to us that contain SHAD and/or Nisat original experimental results is given in section VIII.A. When cited in this report, references in that list are marked with an asterisk after the date [*].

The SHAD experiments, including profiles, were covered briefly in the monograph edited by Miller and published by NOAA (1976*). Adams and associates presented an overview of the SHAD experiments at the Sixth Symposium on Underwater Physiology held in 1975, (1978*), and Dougherty discussed pulmonary function results of SHAD at the same meeting (1978*). A report on CO_2 retention and EGG responses to exercise has recently been submitted by Schaefer et al, (in press). Biochemical results in SHAD III and Nisat I were published by Heyder and colleagues (1979*) and hematology was reported by Murray and Jacey (1977*). ECG changes observed in SHAD and Nisat I were reported by Wilson et al, (1977*). Harvey's observation on the gas switching carried out in Nisat/He II and III were mentioned in an extensive report on the counterdiffusion phenomenon published in Science by D'Aoust and colleagues (1977*).

Two reports cover the SHAD-Nisat effects on vision, VER's, and the EEG--a 1974 NSMRL report (Kinney et al, 1974*) on SHAD, and coverage of Nisat in Undersea Biomedical Research (McKay et al, 1977*). A long report by Weybrew covers the selection of the SHAD I divers (1974*), and another describes the psychological evaluation and methodology used in assessing the diver's response to living in high nitrogen pressures (1978*). Human factors work has been reported by Moeller (1974*; 1976*). A short report by Bondi and colleagues describes how the gas shift was made in Nisat/He (1977*).

Numerous abstracts have also been published and those are included in the list.

F. Progress to the time of this report

For a project stretching over this many years it is appropriate to mention the happenings since the experiments. There were several U.S. habitat-type projects, all sponsored by the National Oceanic and Atmospheric Administration, NOAA. These include the SCORE project involving saturation at 60 fsw and excursions to as deep as 300 fsw, the PRUNE experiments in Puerto Rico, and the continued operation of the Hydro-Lab habitat. Other laboratory projects include the Tonofond experiments of de Lara in Spain, the Airsat series at NSMRL, and a few commercial operations. (Some of these are covered in the monograph by Miller et al, 1976*, and in the NOAA Diving Manual, 1979.)

The SCORE operation began with laboratory runs at Duke University (Miller et al, 1976*). The plan was for saturation on air at 60 fsw, with 60 minute excursions to 200, 250 and 300 fsw. The excursions were longer than those allowed by NOAA OPS for that depth, so were calculated using a slightly less conservative modification of the original criteria. One case of decompression sickness (DCS) was encountered in the laboratory runs following a 300 fsw, 60 min excursion. In the at-sea operation excursions were limited to 250 fsw and 45 minutes (with tables calculated for one hour) and there were no bends reported.

There were several problems with oxygen toxicity, but none were at storage depth. Numb fingertips were noticed by two subjects, once following an excursion, another following a treatment. One subject had an oxygen convulsion after 54 min at 300 fsw on air, and another went into shock (this happened after decompression and was diagnosed as due to dehydration). Later when the project was at sea another subject had a convulsion at 140 fsw while swimming with a recirculating closed circuit rebreather and had to be rescued; the actual cause of this incident was not determined, but it was likely attributable to the apparatus.

Saturation decompression from 60 fsw was conducted using a staged modification of the NOAA OPS II schedule (the original one used linear ascent), without problems. This decompression has been used on other occasions since and is considered reliable for ascent from no deeper than 60 fsw.

Another objective of SCORE was to evaluate the "adaptation to narcosis" observed in NOAA OPS. Test results were equivocal. Subjects operating at 250 fsw felt they were not limited by narcosis, but they did feel it. In terms of the NOAA OPS observations the 50 fsw saturation depth would be expected to be of limited benefit.

Another series of open sea dives were conducted from the La Chalupa habitat in Puerto Rico (Miller et al, 1976*). PRUNE I involved nitrox saturation at 100 fsw with ascending excursions, and PRUNE II saturated at 106 fsw for the purpose of testing descending NOAA OPS excursions. Divers reached depths as deep as 265 fsw, with no decompression or oxygen problems. At 265 fsw one diver terminated his descent because of a feeling of "impending narcosis." Limited performance testing failed to show evidence of "adaptation" to narcosis but subjectively the divers did not perceive narcosis as a problem, at depths normally considered to be too deep for air. Decompression in both 2-week dives was by the Tektite 49-hr profile (NOAA Diving Manual, 1979).

In operation since 1971, the Hydro-Lab program continued, and through 1975 a total of 343 divers had made saturations at 42 fsw from 1 to 13 days (Miller et al, 1976*). NOAA OPS excursions were made to as deep as 200 fsw, and were without problems. Some extended-duration excursions to 200 fsw were also successfully conducted. The Hydro-Lab saturation decompression from 42 fsw has caused no bends except a questionable case in a team of 4 divers while flying after decompression.

A series of experiments known as Tonofond were conducted by de Lara during 1971-75 at the Submarine Medical Laboratory of the Spanish Navy (Centro de Buceo de la Armada, 1976; also reviewed by Miller et al, 1976*). These involved laboratory saturations (0.35 PO_2, balance nitrogen) at 30 and 35 meters of sea water (98 and 114 fsw) with excursions to as deep as 30 msw (261 fsw). Some were shorter, some longer than NOAA OPS but considering their independent origin were remarkably similar. It was felt by de Lara that these aquanauts adapted to habitation completely; they were not bothered by nitrogen narcosis during excursions.

The habitat Helgoland was emplaced at 112 fsw off Maine in 1975 for marine biological (mostly herring) studies, Project FISSH (NOAA Diving Manual, 1979). This project did not use excursions; it received publicity because of a fatality, due, it was felt, to cardiac failure provoked by embolism (Bond, 1975).

The NSMRL followon to SHAD and Nisat is a project called Airsat, begun in 1977. The project is not yet complete, but preliminary data has been reported (Dougherty et al, 1981; Eckenhoff et al, 1981). Airsat I followed the same pattern as SHAD III but with saturation at 60 fsw; Airsat II involved 60 fsw saturation with daily 2-hour excursions to 150 fsw (followed by 2:40 decompression). Airsat III involved both excursion and gas switch. Normoxic (0.3 atm PO_2) saturation in nitrox at 132 fsw with 5-hr air excursions to 198 fsw was followed by a shift to air in the habitat 24 hr before decompression. Airsat results to date have shown decrement in forced vital capacity (FVC) in 26% of the subjects, some substantial. Incidences in I were 1 of 11; II, 3 of 11; III, 6 of 12. All returned to normal but in some divers it took several days. Decrements

were as high as 23% (III). All subjects that did not show a decrease in FVC showed a slight increase. This increase has been noted in numerous other saturation exposures, and is thought to be a combination of improvement in FVC technique and a real effect of the environment.

A control dive at NSMRL (not, unfortunately, called SHAM I) showed this FVC increase in subjects kept in the chamber and not subjected to pressure or PO_2 increase, but it was less than is observed in dives. The Airsat subjects' ability to exercise was minimally reduced. Narcosis was apparent in the excursions, and though there was an improvement in psychomotor capability during habitation, it never returned quite to normal.

The U.K. Department of Energy has initiated a program at the British Navy's AMTE Physiology Laboratory to extend and expand the air saturation-excursion concept and work out the delay necessary after the last excursion before saturation decompression can commence (Hennesey et al, 1981). Wisely they worked out an air saturation (from 15 msw) first, then titrated the delay necessary after a 3-hr excursion to 34 msw before starting saturation decompression. For this excursion using delays in steps of 9 hr they found an 18-hr delay satisfactory; further details will have to await their report.

Documented commercial development of the habitat diving concept has been rather limited, but there have been a number of commercial applications which have not been reported (CIRIA UEG, 1978). Typically saturation depths have been at around 100 fsw or slightly deeper, with excursions to 160 fsw or so. No bends following excursions have been reported but decompression from nitrox saturation is a consistent problem. One documented job used excursions to train welders and included a switch to heliox, necessary for the welding; there were no decompression problems (Peterson et al, 1979).

G. Summary findings and conclusions

The principal findings from the SHAD-Nisat experience are summarized here.

1. Operational overview

Seven dry chamber exposures were conducted over a 3-year period covering a mixed set of objectives, including shallow saturation (50 and 60 fsw) using air with short upward and downward excursions, day-long working excursions to 100 fsw, habitation in a dense narcotic nitrox atmosphere, and a gas switch of saturated divers from nitrox to heliox. A comprehensive monitoring and testing program covered blood and urine chemistry, hematology, physiology and vital signs, pulmonary function, psychological assessment, human factors, oral biology, sensory and brain function, microbiology and Doppler ultrasonic bubble detection.

2. Diver selection criteria

Selection criteria based on characteristics of a normal diver population were used successfully in crew selection for SHAD I. These included congruent personality profiles; similar aptitudes, interests and motivation; no neurotic trends or emotional problems; and relatively more deep diving experience.

3. Shallow excursions

The NOAA OPS excursions with air from air saturation proved quite safe from a decompression point of view. Short descending excursions in SHAD I and II resulted in Doppler-detectable bubbles only one time, but bubbles were heard after all except the first of the 8-hour SHAD III excursions. Some ascending excursions resulted in itching and rashes which resolved on return to saturation depth, and bubbles were detected during a few of these excursions but bubbles and itching were concurrent in only one case.

4. Blood changes from long exposure to hyperbaric air

Living in air at 50 and 60 fsw, with occasional excursions, causes no lasting damage but some adaptive responses. Most striking is the reduction of red blood cells, apparently due to an inhibition of erythropoesis. Extensive blood sampling exacerbated the RBC loss, but control subjects verified that the primary effect was due to the exposure. Parotid gland secretions were reduced, also apparently due to oxygen excess.

5. Deconditioning following long saturation

After the 30 days of saturation in SHAD I both divers suffered from deconditioning and loss of aerobic capacity. There were four exercise periods during the dive; more exercise was scheduled for SHAD II, and these divers were less affected. The SHAD II divers had a greater drop in red blood cells, suggesting that this was not the primary cause.

6. Pulmonary function changes

The long SHAD I and II exposures to PO_2 of 0.52 and 0.58 atm caused no observable changes in pulmonary function except those due to density. Living at 0.52 atm PO_2, one subject in SHAD III had chest pain and showed cyclical reductions in forced vital capacity coincident with daily 8-hour exposures to a PO_2 of 0.84 atm, and another showed a definite reduction in vital capacity following hyperoxic treatment near the end of the dive. Treatment for decompression sickness after Pre-SHAD showed an intolerance to oxygen in both subjects. From a pulmonary point of view residence at a PO_2 of up to 0.58 atm is acceptable, certainly for short periods. Neither CPTD nor COTi dose formulas correctly predicted the toxicity observed.

7. Sickness in 7 atm nitrox

Exposure to a nitrogen-oxygen mixture having an oxygen partial pressure of 0.22 atm and nearly 7 atm of nitrogen caused nausea and vomiting in 2 of 3 divers, starting 2-3 hours after compression and lasting 2 days. The divers were partially relieved within a few hours after oxygen was raised to 0.30 atm. The exact etiology of this sickness is not known; it may have been low oxygen, the high nitrogen, a combination of both or some other unknown cause. Low oxygen in the dense breathing mix was undoubtedly a factor, but the symptoms were not those characteristic of hypoxia; they were not typical of narcosis either.

8. Performance and "adaptation"

Performance tests during the SHAD dives showed minimal evidence of adaptation, either to the extreme depths covered in the excursions or to the saturation itself. Interestingly, however, the scores on performance tests performed during SHAD II (60 fsw saturation) excursions showed less difference from controls than those of SHAD I (50 fsw saturation), perhaps because of the deeper residence depth. Performance was reduced at first by the Nisat I environment of 7 atm nitrox, but returned essentially to normal after 4-5 days. In some tests there was a secondary drop in performance scores after 3-4 days. Nisat I divers felt quite "drunk" the first few days, and were aware of the narcosis until the first half of decompression was complete.

9. Heart rate decrease

The resting heart rate of the SHAD and Nisat I dives showed a decrease on compression followed by a gradual (8-day) return to normal. Rate decreased further during descending excursions and speeded up again on the short ascents and during decompression. Heart rate rose prominently postdive to SHAD I and II. These are felt to be changes in autonomic control of the heart, and though it involves oxygen, pressure, and gas density, the exact etiology is unknown. Extensive monitoring of EKG parameters failed to disclose any deleterious effects on the heart detectable by these methods; there were deviations during periods of nausea. One Nisat I subject showed disappearance of the p wave and arrhythmia during heavy exercise, causing his exercise to be discontinued.

10. Visual function and EEG

Visual effects were slight and included a predictable shrinkage of retinal vessels in the hyperoxic exposures. Changes in evoked responses were typical of air diving. Some EEG changes, though slight, could not be attributed to the known stresses. These resemble generalized CNS dysfunction with no identified cause. Recovery was complete and immediate on decompression.

11. Counterdiffusion following gas switch

Switching the inert background gas in saturated divers from nitrogen to helium with the oxygen level unchanged provoked problems. In saturation at 66 fsw this caused itching 3 hours after the shift, lasting 5-6 hours. At 99 fsw itching began within 1 hour and caused rash and welts predominately on covered areas of the body; it lasted 5 hours and was intense. The itching was promptly relieved by shielding the diver from the helium environment. Eight hours after the switch one diver got classical bends in both knees which responded to conventional treatment by recompression. An economical method was devised to make the gas switch, saving both gas and time.

12. Saturation decompression and effect of excursions

The use of the 480 minute halftime tissue compartment was found to be unsatisfactory for nitrox saturation decompression. Long excursions (e.g., 8 hours) that do not cause overt decompression sickness apparently created, through gas loading and/or bubble formation, a decompression obligation that required more than a few hours to clear. Decompression sickness occurring during decompression from saturation after SHAD III seemed to be related to the 8-hour excursion completed 16 hours before. A conservative decompression from the heavy 7-atm nitrogen load in Nisat I was without problems.

H. Photos

Figures 1-1 to 1-12 were taken at the time of the dives. They show the facility, personnel, and some of the experimental apparatus. All photos were made by the NSMRL photo lab; Figure I-1 appeared in U.S. Navy Medicine (Adams, 1974*).

Figure 1-1. Topside crew and investigators during SHAD I.

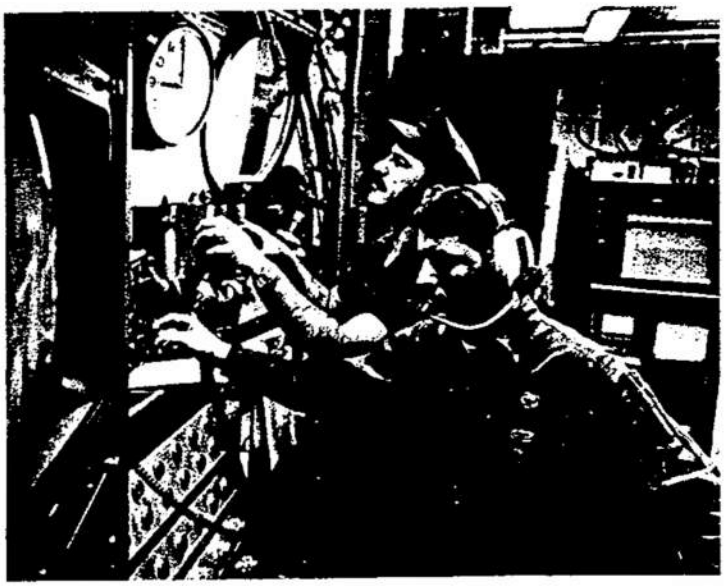


Figure 1-2. SHAD I Divers WB and JW working at controls in SHAD II.

Figure 1-3. Diver GS placing electrodes on RF during SHAD II.

Figure 1-4. Awaiting the surfacing of SHAD II.

Figure 1-5. Overall view of chamber area during SHAD II.

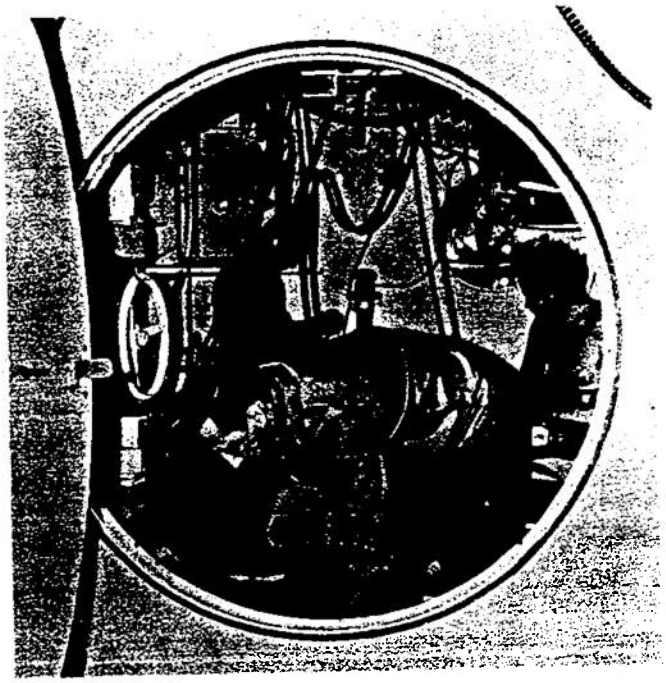


Figure 1-6. Nisat I crew, JB in foreground, with NT (1) and RJ.

Figure 1-7. Nisat/He II divers taking pre-dive saliva samples. Left to right MH, RO and JC.

Figure 1-8. Diver RO doing pulmonary function, Nisat/He II.

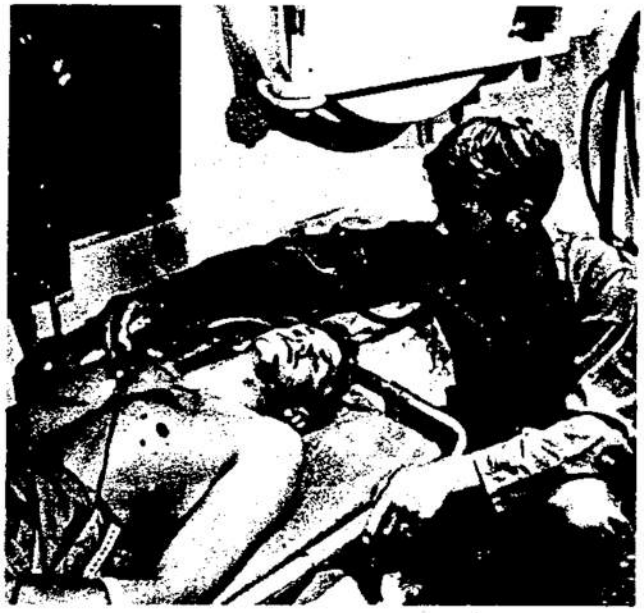


Figure 1-9. Diver RE adjusting ECG on RL, Nisat/He III.

Figure 1-10. Topside Doppler monitoring, Nisat/He III.

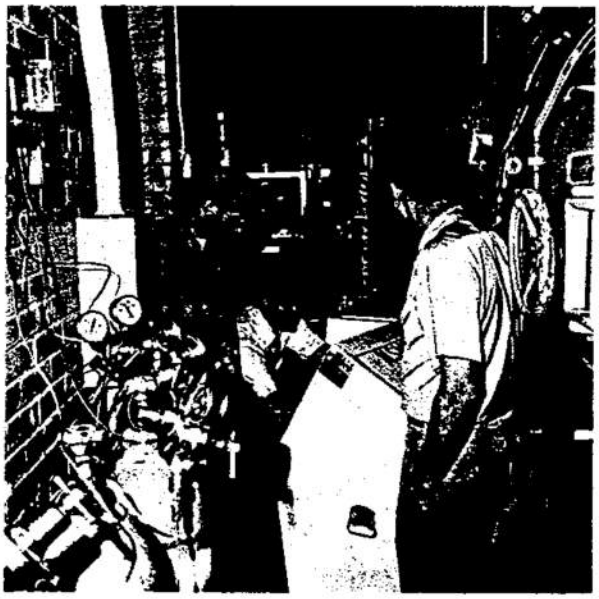


Figure 1-11. Adjusting mass spectrometer in area behind chamber.

Figure 1-12. Some of the topside crew during SHAD III.

II.
GENERAL EXPERIMENTAL SETUP

R.W. Hamilton and J.E. Jordan

This chapter covers most of the consistent functional details involved in conducting the SHAD and Nisat experiments, including facilities, operators, schedules, subjects, safety, and specialized laboratories.

The experiments were conducted over a 3 year period during which some changes were made in both procedures and the facility. However, most functions were the same throughout the series, and these are discussed in this chapter. To a certain extent the use of past tense in the discussion about the facility helps identify functions that are no longer the way they were at the time of the experiments. Any special procedures and environmental differences significant enough to have an impact on either the divers' environment or experimental results are covered with the individual experiment.

A. Facility

All exposures were performed in the hyperbaric facilities of the Environmental Simulation Branch, NSMRL. The facility occupies two large rooms of Medical Research Laboratory Building #141 and some outside space. The facility is capable of conducting non-saturation (bounce), saturation and excursion diving with either air or mixed gas, in the range to 350 fsw.

The pump room houses heavy machinery and equipment such as compressors, heaters, chillers and water pumps. The chamber room contains two hyperbaric chambers designated as 1 and 2, their associated equipment, a briefing and conference area and a small kitchen alcove (stove, sink, refrigerator, etc.). A water cooling tower for the compressors and gas storage equipment are outside the building. The layout of the laboratory is shown in Figure 2-1. This figure was taken, with modifications, from a manual of the hyperbaric complex prepared by Potomac Research, Inc. (1981). Figures 1-1 and 1-5 show the chamber area.

1. Chamber system

a. Physical characteristics

Chamber 1, also known as the Genesis chamber, is a horizontally oriented cylindrical steel pressure vessel 25 feet long and 9 feet in diameter. It is divided into two independently controllable locks, designated "inner" and "outer;" their volumes are 1000 and 700 cubic feet.

The useable length of the outer lock is 8 feet; it serves as the entrance way into the chamber. A 48 inch exterior hatch (door) swings inward and seals under pressure when the lock is pressurized. A 48 inch inner hatch also swings into the outer lock and makes it possible for the outer lock to be pressurized independently of the inner lock. There is a single large viewport between the locks.

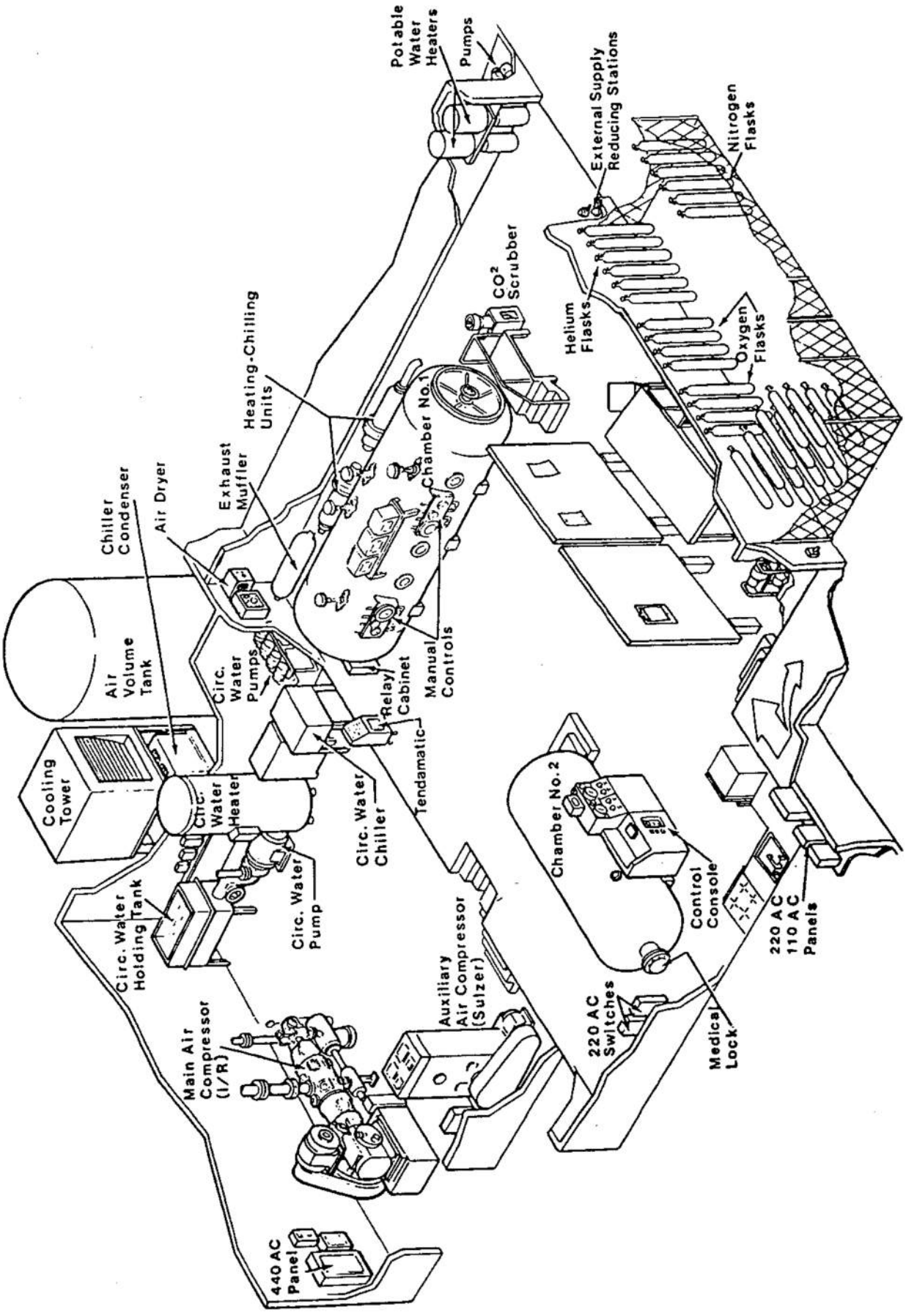


Figure 2-1. Overview of NSMRL hyperbaric facility. This sketch was taken from the manual prepared by Potomac Research in 1980. It has been modified to delete items dated since SHAD-Nisat (1973-1976) and to include things that were there then but have been removed.

The outer lock was equipped with:

gas supply manifold
built-in breathing system (BIBS)
overboard dump manifold
shower and sink
2-way speaker for diver communication system
headset junction boxes
depth gauge (caisson gauge, provides internal pressure)
physiological monitoring penetrations
fire hose
oxygen sensor
storage locker
single exterior viewport
TV camera (inside)
emergency lighting
sound powered phone

The useable length of the inner lock is 13 feet; it is entered from the outer lock. The inner lock was equipped with:

gas supply manifold
built-in breathing system
overboard dump manifold
food and medical transfer lock
2 bunks
2 TV cameras (inside)
physiological monitoring penetrations
3 shelves
2 storage lockers
2 exterior 13" viewports
2-way speaker for diver communication
headset junction box
sound powered phone
entertainment speaker
fire extinguisher
emergency lighting
oxygen sensor
depth gauge (caisson gauge, reads internal pressure)
life support system
nitrogen purge system, for electrically powered instruments

A food or transfer lock (often called the "medical" lock by divers) of 30 x 18 inch internal diameter built into the wall of the inner lock enables the transport of small items between the hyperbaric chamber under pressure and the outside. Transfer with this lock can be accomplished quickly if necessary, or alternately it can be decompressed at a slower controlled rate by employing the flowmeter installed on the vent line.

During SHAD and Nisat the chambers were operated with a set of controls located at the side of Chamber 1 (Fig. 1-3). Controls for each lock included 2 pressure gauges (primary and backup), valves for pressurization and decompression, and a communications terminal. Since SHAD-Nisat this has been replaced by a remote console about 15 feet away, but it still serves a backup direct-control function.

b. Air supply system

Compressed air is stored in a 1000 cu ft 350 psi volume tank located outside the facility. The pressure in this tank is displayed on several gauges in the chamber room.

The facility has two compressors for chamber pressurization: one main and one auxiliary. The main compressor is a 1966 Ingersoll-Rand model ESH2NL2 horizontal non-lubricated teflon ring compressor with a discharge pressure of 350 psi and a suction volume of 158 scfm. It is powered by a 50 hp electric motor. It is equipped with an Ingersoll Rand type TD-100 Tendamatic control which serves as a remote, centralized, control and monitoring station for the compressor and as an operating station for the auxiliary compressor.

The auxiliary compressor is a Sulzer model 2E130-2A, labyrinthine ring, non-lubricated compressor which generates a pressure of 350 psi and a suction volume of 160 cfm. It is driven by a 70 HP electric motor.

Compressed air from both the main and auxiliary compressors is delivered to the large volume tank.

c. Gas supply

For the SHAD dives only air was needed; Nisat required in addition both nitrogen and helium. The nitrogen "pure" gas supply was stored in a tube trailer (approx. 40,000 cubic feet) located just outside the building. Gas pressure was reduced at the trailer to 375 psig with a dome loading regulator, and the gas was piped to the chamber via a high pressure hose equipped with a relief valve. The helium gas supply was also stored in a tube trailer and handled in the same way. During use the individual tubes were opened one at a time ("cascaded") to maintain maximum available pressure in the unused gas. The tube trailers also served as a backup "air" supply in the event both compressors were not functioning; by addition of oxygen a "synthetic air" mix could be supplied.

Pure oxygen was introduced directly into the chamber for maintenance of the required oxygen partial pressures, ("metabolic" O_2; see g, below) and for the BIBS manifold (section f). Oxygen is stored outdoors and is reduced to 90-125 psig before it enters the building.

Other breathing gases (except air) used on the BIBS system were supplied from cylinders and connected when needed.

d. Pressurization and decompression

Both chambers are designed to operate with a pressure range from 0 to 156 psig or a simulated depth of 0-350 feet of sea water. The main lock can be pressurized at a rate of up to 75 fsw/min. Decompression can be at up to 60 fsw/min when deeper than about 60 fsw. Decompression from 60 fsw to the surface takes 3 1/2 to 4 min; from 10 fsw, 1 1/2 to 2 min. Pressurization rate for the Nisat I dive (the deepest one in the series) was limited by flow from the tank truck; it took 51 minutes to reach 198 fsw.

Manually controlled valves on the chamber were used to control gas flow into and out of each lock, separately or together.

e. Life support system

During SHAD-Nisat a life support system controlled and monitored the atmosphere for the inner lock of the Genesis chamber. This system uses conventional methods to remove CO_2 and humidity and adjust the temperature of the chamber gas. Gas is circulated through an external loop. On leaving the chamber, still at chamber pressure, the gas passes through a cannister filled with high moisture (medical grade) Sodasorb (W.R. Grace and Co.) for CO_2 absorption. The gas then passes through a heat exchanger supplied with cold water where humidity is removed by condensation, followed by a warm water heat exchanger to reheat the gas. A blower then forces the gas back in at the top of the chamber; normal flow is 90 acfm.

The temperature of both the dehumidifier and reheater coils was controlled manually by adjusting the water flow rate, or with a timer. Condensate from the cold coil drained into a collecting tank which was periodically emptied. The blower is a squirrel cage type that has a "canned" motor which is considered fire safe. Humidity was monitored, but diver comfort was the main determinant of the humidity control.

The CO_2 absorption cannisters hold about 50 pounds of Sodasorb and have to be changed every 4 man-days of chamber occupancy or every 24-36 hours. The life support loop can be isolated for servicing.

Odors were controlled by the addition of Purafil (activated alumina pellets impregnated with the oxidant potassium permanganate) to the CO_2 scrubber cannisters.

Since SHAD-Nisat a scrubber has been added to the life support system of the outer lock, and automatic controls have been installed for the dehumidification and heating coils.

f. Built-in breathing system and overboard dump system

The BIBS (built-in breathing system) provides a means of delivering breathing gas to chamber occupants by mask. It is used to deliver selected gases (oxygen, air, heliox, nitrox) during treatment, experimentation, gas switching or emergency operations.

The BIBS system consists of gas supply manifolds running the length of the top of the chamber on either side. Hansen quick disconnect outlets are located overhead in the center of the chamber about every 2 feet. The users wear Scott 800238-01 series divers' inhalators (oronasal masks) equipped with demand type supply and exhaust regulators.

Exhaled gas from the exhaust regulator on the face mask goes to an "overboard dump" manifold. It is then piped through the chamber shell and into a collection tank equipped with a Moore model 63BU constant differential flow controller. The flow controllers (one for each tank) are referenced to the lock's ambient pressure and maintain a constant 3 psi negative pressure on the overboard dump line.

g. Oxygen makeup

Within each lock is an oxygen probe containing a primary and backup fuel cell type detectors. Signals from these are sent to two Teledyne model 323D solid state oxygen controllers and readout meters; these controllers were installed after SHAD II. These add O_2 through a solenoid-controlled 1/4" line into the suction line of the life support system as it leaves the chamber. During SHAD oxygen was added manually, and it is still done this way on occasion. When needed pure oxygen is added directly into the chamber. The operators add oxygen for 20-30 seconds, then wait for a reading in order to avoid overshooting.

h. Electrical system

Main power to the hyperbaric facility includes 440 VAC, 220 VAC, 110 VAC and 6 VDC systems; the DC power is from a Hewlett-Packard (EQ6) variable DC power supply unit. Emergency power sources include a 220 VAC portable generator and three 12 VDC batteries.

In the event of main power failure during chamber operation, the facility can be switched to a standby emergency generator. It is a 60 cycle, 60 KW, 3-phase 220 VAC generator driven by a portable diesel engine and is capable of powering the complete complex, except the air compressors. Other procedures taken during a power failure are described in the safety section, II.B.6.

i. Communications

Communication between the divers in the chamber and topside personnel in the chamber room was by means of an audio amplifier using headsets, a Marvel Dive-Com system, and a sound powered phone.

The principal method for audio communication was the headset system; using headsets with boom mounted microphones. Four channels of headset communication were available for isolated or semi-isolated discussions between the individual divers and various topside personnel.

The Marvel dive communication system is essentially a two way intercom system. The divers talk into a transceiver box from which the sound signals are transmitted to a transceiver in the chamber room. The divers do not press a switch to talk or listen, but a switch must be pressed by the topside operator when talking into the chamber. The transceiver in the chamber room is usually turned off temporarily during pressure changes to reduce noise.

These systems were effective under routine conditions but at times, such as during excursion pressure changes, etc., were intermittent and inadequate. A new multi-channel system was installed after Nisat.

The sound-powered phone looks like a standard household phone but uses no external electrical supply. The power to the receiving set is generated by the speaker's voice acting on a piezoelectric microphone. The signals are low level and the phones are considered fire safe.

For SHAD I and II the video system consisted of a Hydro Products model 100 helium-proof underwater television camera installed in the inner lock. An additional TC 125RA camera was placed in the inner lock after Nisat I, and one was installed in the outer lock and all major areas of that chamber can now be visualized. Visual display is on 3 Conrac model SNA9 17" monitors mounted on a shelf above the chamber control console.

An AM-FM radio and hi-fi tape player was patched into the intercom. Movies used this amplifier and speaker for sound while the movie was projected into the chamber via a viewport.

In the event of total failure of electronic communications and the sound powered phones, mallets were kept on board for rudimentary communication using the standard diver/tender signals.

j. Lighting

Canty lights provide all normal lighting with minimal heat and without the need for electrical power in the chamber. Canty lights have high intensity lamps in fan cooled housings on the outside of the chamber wall and transmit light into the chamber through transparent solid acrylic "light pipes" that pass through conventional pressure fittings and seal with pressure. Each lock can be individually controlled for luminous intensity. Chamber 1 has eleven Canty lights, eight in the inner lock and three in the outer. This is sufficient for ambient-light photography.

The emergency generator provides backup power to the Canty lights. A battery powered emergency lighting system located above the chamber room entry is automatically activated during a power failure and 12V bulbs provide emergency chamber lighting. As a further backup a battle lantern is kept in the chamber room.

k. Personal hygiene system

A wash basin and shower with hot and cold water is available in the outer lock. The water is drained into a covered bilge collecting pan in the outer lock and periodically purged into an external drain. Domestic water is pumped by a Hydro model 5200 electrically driven pump to the shower, sink and fire suppression hose inside the chamber. The pump is needed when the chamber is pressurized in excess of 60 fsw; it is turned on and off by the operator. Check valves prevent chamber pressure from entering the building system.

Prior to SHAD II solid waste was collected in plastic bags installed in a portable stainless steel commode ("camp stool") in the outer lock. Urine was collected in plastic jugs. A portable toilet (without the chemical additives) was used in SHAD III and subsequent dives; it was locked in and out when needed and was serviced by topside personnel.

l. Fire suppression system

The fire suppression system in each chamber has two components: a Navy-type stainless steel fire extinguisher containing water and overpressurized, and a fire hose connected to a cold water line. The same piston pump used for personal hygiene water provides water to the fire hose. There is no deluge system.

m. Nitrogen purge system

The nitrogen purge system consists of two nitrogen flasks and a gauged and valved delivery line which enters the rear of the chamber. The system is used for all scientific equipment that uses 110 volt power within the chamber. In operation, nitrogen is purged into the case of the instrument at a flow of 1/2 liter/min. This surrounds electrical components to prevent or reduce the fire hazard due to raised oxygen.

2. Backup or standby chamber

The backup chamber or Chamber 2 is kept in readiness for treatment of decompression sickness or embolism whenever the main chamber is used for human dives. It has an internal diameter of 6.5 feet and an overall length of approximately 15 feet. It has dual locks (inner and outer), and is capable of pressurization to a depth of 350 fsw (156 psig) at a rate of 100 fsw per minute.

The outer lock is 4 feet long and the inner lock is 10'7" long, and there is a transfer lock in the far end of the inner lock.

Chamber 2 is primarily designed for short duration dives. Unlike chamber 1, it has no heating or chilling equipment for temperature and humidity control, but can be controlled by a combination of venting and room temperature control. It is however, equipped with a BIBS (Built in Breathing System), with overboard dump capability, and an external loop CO_2

scrubber (installed 1974). It is suitable for any USN treatment procedure or for any of the special ones which might have been needed in the SHAD and Nisat series.

During SHAD I and II the backup chamber was the chamber at the Submarine Escape Training Tower. Transfer to that chamber was rehearsed and could be accomplished in under 10 minutes (see II.D.11.a).

3. Monitoring

Chamber 1 is equipped with about 50 multi-wire electrical penetrations containing over 200 leads for physiological and other monitoring equipment. These are used for EKG, Doppler ultrasonic bubble detector, stabilimeter, ergometer, spirometer, and a variety of other instruments and equipment. A single 32-wire penetration (Lundy) was used exclusively for EEG signals.

a. Timing

A primary digital clock was used for the log and as a reference for other timing devices. It was checked daily by radio and corrected whenever necessary.

b. Pressure

Large (16") Heise pressure gauges of the Bourdon tube type display chamber pressure at the operator's station; these were used throughout SHAD-Nisat for primary pressure control. Following Navy tradition a lighted magnifier permitted the operator to maintain the needle to within a few inches of sea water; pressure rarely varied throughout the series. Saturation decompression is conducted in 1/4 fsw (3") steps.

Mercury and water manometers are used for pressure measurement when chamber pressure approaches atmospheric.

The pressure unit used for this report is the foot of sea water, fsw. In normal usage the foot of sea water is defined as 1/33 standard atmosphere or 23.03 mmHg or 0.445 psi. The calibration used for the Heise gauges is presumed to be this.

The gauges are calibrated by the base instrument shop at regular 6-month intervals.

c. Oxygen analysis

A Beckman F3 paramagnetic analyzer was used as the primary oxygen monitoring system. Chamber gas was reduced to atmospheric pressure, dried with silica gel, and passed directly into the oxygen analyzer. Oxygen percentages recorded on the working logs were read directly from the F-3 meter. These were converted to partial pressures for the logs included in the appendix. A Teledyne Model 323D oxygen controller was used to control oxygen in the chamber and as a secondary oxygen monitor.

The Medspect mass spectrometer (Scientific Instruments Model MD-8) used for respiratory studies served as an additional back up monitor for oxygen and CO_2.

Also backing up the primary monitors and available for trace gas analysis was a Hewlett-Packard Model 7620B gas chromatograph with Por-a-pak Q columns and sub-ambient temperature programming capabilities.

d. Carbon dioxide analysis

Chamber carbon dioxide levels were monitored by means of non-dispersive infrared analysis using a Beckman IR-315 having a range of 0 to 1% full scale. This instrument is sensitive enough to deal easily with the chamber gas after it has been reduced to sea level. CO_2 readings were entered in the working logs as percentages but are shown in this report as partial pressure.

Carbon monoxide was periodically checked by introducing spot samples into an Ecolyzer CO analyzer.

e. Humidity and temperature

Humidity was monitored by a conventional hygrometer in the inner lock (Abbeon AB-167) during SHAD, later changed to a Hygrocon impedance type humidity sensor. The detector element of the latter instrument uses a plastic wafer that has on it a conductive layer separated by a chemically treated polystyrene which changes its impedance as it absorbs moisture. The change in impedance (resistance) is measured by a bridge circuit and displayed on a meter. This sensor was not reliable in our hands, and RH readings were occasionally not available.

At times a Dwyer wet bulb/dry bulb thermometer was used. Diver comfort was the primary factor in setting the humidity level.

Temperature was read from the monitor of a Honeywell 12 channel controller/recorder using copper-constantan thermocouples. In addition Yellow Springs thermistors were used.

B. Operations

The operational aspects of the project were planned in some detail in advance, and from a safety point of view it was treated as a high risk exposure.

1. Crew staffing

Total responsibility for the maintenance and operation of the chamber facility (Environmental Simulation Branch) during the SHAD-Nisat series rested on the Facility Supervisor, a highly experienced retired USN Master Diver.

The chamber crew consisted of a minimum of 5 people on each shift, with one extra during the day and occasionally an extra during the evening shift. Shifts were the classical ones, 0800-1600, 1600-midnight, midnight to 0800. Crew positions were supervisor, operator, recorder, gas analyst and messenger. When an additional person was on duty he had the duty of second operator. Also, a regular cook worked during SHAD I and II. "Watch bills" for each dive were prepared in advance, and changes were arranged as needed. Chamber operators and analysts were hospital corpsmen trained at NSMRL for hyperbaric nursing or qualified by examination; diving experience was not required. Recorders and messengers were enlisted personnel, with no special training requirements. Watch standers filled out the physical examination forms for the person doing the exam, handled urine volume measurement and sample handling, ran EEG's, etc.

The Supervisor was trained to operate every facet of the facility. Supervisors were always ex-USN divers (retired or active) with considerable experience. There were no specific criteria for Supervisors, but they were trained and qualified on the facility, then personally selected by the Facility Supervisor.

In addition to these shift jobs certain duties were assigned during the course of a dive. These were project coordinator and assistant, experiment scheduling coordinator, watch scheduling and training, medical supervisor, food procurement, purchasing and supply, financial recording, communications and engineering, diving supervision, and movie procurement. Some people did more than one of these jobs. These jobs were in addition to the crew duties for many individuals.

A medical officer was present in the chamber room or in the building 24 hours a day whenever divers were at pressure; his location was always known by the shift Supervisor. More detail on medical backup is given in section II.D.

For most of the scientific measurement procedures one or more investigators were present during data gathering periods.

A "pass down log" was maintained for items which were of interest or had to be taken care of by a succeeding shift. This was used for medical procedures, but also for operational discrepancies.

2. Diver personal requirements

To minimize discontent of the subjects the topside crew was, generally, quite responsive to personal needs.

a. Food

During SHAD I and II a temporary culinary specialist was employed to prepare hot breakfast and dinner for the divers. Lunches and snacks were from a cold chest in the chamber which was refurbished each evening. Each SHAD diver was supposed to maintain a log of his food intake with respect to time of day, type, and quantity, and all divers were required to keep fluid intake logs. Certain foods were prohibited because of biochemical tests. The SHAD divers were not allowed to smoke and were limited to 2 ounces of alcohol per day during the pre- and post-dive periods.

For subsequent dives food was prepared at the base hospital, delivered to the chamber area on trays and rewarmed if necessary in a microwave oven. Comments about the food's quality or taste are rarely noted in the logs, and leftovers were seldom returned to the surface. An effort was made to provide good meals, and this was regarded as an important morale factor during the long confinements.

In later dives the person responsible for food was also charged with maintaining proper food sanitation according to Navy preventive medical procedures (U.S. Navy Medical Dept., 1972).

b. Hygiene

In the early part of the program (SHAD I) waste bags from the camp stool had to be handled through the small medical lock. The bags of course were vented before being decompressed. There were a few minor mishaps in this routine but these were without further consequences. In modern commerical saturation diving this handling of waste, particularly feces, is considered unacceptable because of the increased liklihood of external ear infection. Later in the series a chemical toilet was left in the lock and it was not necessary for the divers to deal with it. Urine was collected for measurement and analysis so was passed out immediately.

The shower and lavatory facilities worked well and though the divers were not especially pleased with the arrangements they were regarded as primitive but acceptable. Each diver was able (and expected) to shower each evening.

Periodically during the long dives a crew of two technicians medically qualified for pressure exposure were locked in to scrub the chamber's inside surface with soap and water. The divers performed a "field day" (cleanup) on their lock twice weekly.

Divers wore loose fitting coveralls made of fire resistant material, regular underwear, and sneakers or slippers.

c. Entertainment

"Full length" movies were shown almost every night and were an important morale factor. These were obtained through normal government channels on a regular basis and followed a schedule. The projector was set up to project through a porthole onto a screen inside, where the divers listened on speakers installed for the purpose.

Hi-fi music, news, and entertainment was also available from recordings or local radio, and newspapers and magazines were locked in daily.

The main source of entertainment was the old standby, conversation; among the divers, between divers and topside, with visitors qualified to enter the chamber, and otherwise over the communication system. Telephone calls could be conducted also.

d. Sleep

Subjects slept between 6 and 7 hours each night. Lights were out usually by 2300 hours and divers were awakened at 0600 or soon after. Some divers napped frequently an hour or two during the afternoon when the schedule permitted. Comments were recorded in the logs regarding drowziness, however, no complaints were made or insinuated about insufficient sleep time or inadequate sleeping quarters. The logs reflect that on occasion testing procedures delayed bedtime. When 3 divers were in the chamber one of them had to sleep on a mattress on the floor of the chamber. There was no in-chamber "watch;" all divers were allowed to sleep all night.

e. Exercise

During SHAD I exercise on the bicycle ergometer was performed infrequently. In SHAD II and III it was more frequent, as it also was in Nisat I, but at a lower level due to the increased gas density. Regular exercise was not part of the Nisat/He II and III protocols. Exercise is discussed in section IV.B.

3. Operating procedures

a. Pressurization gases

For taking the chamber to pressure with other than air procedures were used that avoided the need for premixed gases. In Nisat I the inital pressurization was with pure inert gas, thus maintaining the partial pressure of oxygen that was in the chamber to start with. For the inital pressurizations in Nisat/He II and III the chamber was taken to 15 fsw with air, equipment and analyzers were checked, then pressurization proceeded with inert gas as just described. Filling the chamber with helium for the

isobaric switch experiments is covered in section 5, below. During pressurizations with inert gas the divers breathed air by mask, and they removed the masks in turn.

b. Lock operations

To conserve gas the lock transits were to a large extent planned in advance. The equipment needed for a given set of experiments coming up were loaded into the outer lock usually well in advance of the measurement period, and meals and hygiene supplies occasionally used the same lock trip. In SHAD some supplies were sent down with the daily medical visit. For equipment only the lock traveled in "Mode I," to 45 fsw in 1 min, then slowly down as temperature equilibrated. For unscheduled transfers usually the small "medical" lock was used. The outer lock was kept at the surface during excursions to facilitate transfer of the doctor if a need should arise. Blood samples were decompressed at 8 fsw/min using the medical lock.

c. Visitors

Visitors were allowed to enter the chamber only if they were qualified as USN divers, medical officers, or hyperbaric medical tendors, and then only on no-decompression dives (except for the daily visits in SHAD of the Duty Medical Officer). No visitors entered the chamber on the mixed-gas dives.

4. Operational logs

During all pressure operations a log recorder kept a dive log following traditional Navy practice. Environmental parameters were recorded every half hour and as required by events. These logs followed the format developed at the University of Pennsylvania to make them computer readable. Condensed logs of the SHAD-Nisat dives are reproduced in Appendix A. Time lines of the individual dives are given in section G of this chapter.

5. Isobaric gas switching

In Nisat/He II and III it was planned to switch the divers' gaseous environment as quickly as possible from nitrox (nitrogen-oxygen) to heliox (helium-oxygen), at constant pressure and leaving the oxygen level unchanged.

In Nisat/He II the approach was to have the divers crawl through a zipped plastic diaphragm. The diaphragm was a plastic sheet attached to a wire and plastic hoop which fit into the "dogging" groove in the passageway. The divers were isolated in the nitrox in the outer lock while the inner lock was vented to 10 fsw and its atmosphere flushed with helium. When the nitrogen level was below 1% the inner lock was refilled with helium and the oxygen level was adjusted to 10% and mixed by means of the life support system blowers. Once mixing was complete one of the divers (JC) was to climb through the diaphragm and resecure the zipper.

As it turned out, one of the dogs on the hatch cover displaced the diaphragm and as soon as the hatch was opened to make the transfer the two atmospheres began to mix. The hatch was closed immediately, and after a brief conference diver JC went through into the inner lock and resealed the hoop. As soon as the plethysmograph was ready the next diver (MH) went through. The remaining diver breathed 90-10 nitrogen-oxygen by mask until time for him to transfer.

The log shows that 17% helium was in the outer lock and 17% nitrogen was recorded from the inner lock. These are "worst case" readings, from near the top and bottom of the chamber, respectively. Although the exact mixtures present at this time are not known, it is certain that the effective values were less than 17%, probably in the range of 5-12%. As soon as the first diver was transferred a purge was begun in both locks. A slow vent of pure helium with the oxygen controller adding oxygen enabled the inner lock to meet the "<1%" specification by the time the 3rd diver locked in. The outer lock was also flushed and a partial correction was made there as well.

For Nisat/He III a different approach was used. The divers were dressed in "Nucon" polyethylene suits consisting of hood, jacket, trousers and boots. The suits were modifed so a pure nitrogen purge could be maintained around the divers' bodies. The divers used the BIBS system under the hood, breathing a mixture of 95% nitrogen, 5% oxygen during the flushing process. The inner lock was isolated by closing the inner lock door and the purging of the 1000 cubic foot chamber began with the divers seated in their suits in full view of the TV monitors.

Purging of the inner lock took advantage of the density difference between the two gases; we managed to make the shift in 55 minutes instead of the calculated 117 minutes had the gases been allowed to mix, and used only 8000 cubic feet of gas instead of the 17,000 that would otherwise have been needed. The technique has been described by Bondi (1977*). Gas was bled through a manifold that distributed it along the top of the chamber, and the inflow was kept low enough (148 ft^3/min) to avoid excessive mixing. The life support system was turned off and the divers remained as still as possible except for one who was assigned the task of placing the sampling cannula as directed without moving from his seat. Sampling through the mass spectrometer was used to monitor the level of the interface between the pure helium and the nitrox mixture (though a helium-filled balloon would do perhaps as well). The results were intermediate between the worse case of complete mixing, and the ideal conditions of perfect layering in which case the new gas required would have been only that needed to fill the chamber.

Once the chamber gas reached less than 1% nitrogen the flushing was stopped, and oxygen was introduced and helium released until a mixture of 93% helium 7% oxygen was reached with the life support system on line.

The divers then removed their suits and masks in turn at intervals of about one hour.

6. Safety and emergency procedures

Many aspects of the design of both the equipment and the experiments are safety related and it is not practical to single them out here for comment. Specific areas worth special mention are fire, pressure, and oxygen safety, and dealing with medical problems related to decompression and oxygen toxicity.

During the pressure phase of a dive three distinct aspects were recognized -- operational, medical and investigative. A "supervisor" was designated for each of these, and he had the privilege of making a unilateral decision to terminate the dive due to an unreasonable problem in his area. The decision of one supervisor to terminate could not be overruled by the others, but each was not allowed to base his decisions on conditions in one of the other areas. There were plenty of discussions but none of the dives was terminated.

a. Fire safety

The first aspect of fire safety was an analysis of the degree of risk. All of the SHAD experiments used air as the chamber atmosphere, and compressed air supports combustion better than does air at sea level so precautions were necessary throughout the SHAD series (Dorr and Schreiner, 1969). The Nisat experiments involved safe atmospheres throughout the bottom time up to the time the chamber atmosphere reached 21%; after this time and during oxygen breathing fire precautions were intensified.

Certain items were restricted from the chamber: Matches, lighters, flames, "unapproved" electrical gear (approving authority was the chief of Bio-Medical Engineering and the Facility Supervisor), volatile flammable fluids, unnecessary papers. Divers wore fire-retardant clothing during fire-risk parts of the dives, and sneakers or slippers. Linens were fire retardant. Necessary papers were kept stowed, away from lights and electrical equipment.

In the event of fire the person observing it should report, "Fire in the chamber!" Topside is to secure (turn off) in-chamber electrical equipment and activate the pumps to supply pressure to the water hose. Divers don masks (supplied with air) immediately, and use the hose or extinguisher to fight the fire or move to the outer lock. Air or another suitable breathing gas was supplied to the BIBS at all times, under control of the divers. A bucket of sand was also kept in the chamber.

b. Pressure safety

Pressure safety is largely a matter of system design, and in that regard no special precautions were taken for these dives. One reason for this is that pressures used were relatively low and well below system limits, and failure was somewhat unlikely. Further, the consequences of pressure loss were regarded as manageable (see II.D.11).

c. Building fire or loss of power

In the event of a power loss or fire serious enough to cause termination of the dive, the divers were to be transferred to another chamber, either the escape training tank or Chamber 2. This transfer is covered in II.D.11.a.

C. Diver subjects

All subjects in the SHAD/Nisat program were volunteer naval personnel. Their selection process and all the activities performed were consistent with the role of volunteer experimental subjects. Despite this and the fact that none of them actually got wet at all during the entire program, we have chosen to refer to them as "divers" throughout this report.

1. Selection and screening

It was not easy to find divers for these experiments. We felt they had to meet certain requirements, and coupled with the long isolation and the anticipated risk and discomfort, it was necessary to go outside of NSMRL to secure volunteers. The different dives had different exposure profiles and different objectives, so slightly different criteria were primary in subject selection.

The objectives of Pre-SHAD were mainly to ensure the habitability of the chamber and to "verify" the saturation decompression procedures. We felt that an in-house team would be best so we used an investigator (GA) and a diving corpsman from the Naval Undersea Medical Institute (TT).

For SHAD we used as a subject population base the USN First Class and Saturation divers available from naval installations in the vicinity of NSMRL. Five volunteers were brought to the lab two weeks before the scheduled start of the dive. These were given intensive preliminary testing on the first day, including pulmonary function evaluation, psychological testing and long bone radiographs. During this period the demeanor of the divers was observed by the scientists and reviewed with a committee of responsible medical officers. Any health problems which would preclude effective participation as a subject were evaluated by the committee. In addition to the guidelines in the USN Manual of the Medical Department (1972), any disorders which could significantly impair the interpretation of the many physiological, biochemical, and psychomotor tests were considered disqualifying. A subject selection committee reviewed these data and tentatively chose two subjects to participate in SHAD I, and two others to participate as controls. The fifth was sent home.

The final choice for SHAD I was not made until the day the dive began, when the two subjects whose health, proficiency and psychological status seemed best were selected (WB and SW). The other two (GS and PD, later PP) served as control subjects and stood watch.

The selection of the two SHAD I divers is covered in the report by Weybrew (1974). The selection of the divers was a study in itself, not only for SHAD but to help provide guidelines for other projects of this nature. The divers were given several tests, including the MMPI, a depression-proneness test and a biographical inventory, and were interviewed in detail before and after the dive. There was little specific information available on which to base a selection, so the test and interview results were compared with "normative" information as well as with the other divers in the group. Several groups of Navy divers examined for various other projects were used as a basis, so that the subjects should have personality and motivational characteristics typical of the diver population and a similar set of aspirations and standards of performance. That is, since we could not say exactly what makes a good diver, the study used as a standard the characteristics of (presumably) successful divers and what they thought a good diver should be. The two divers who served as controls for SHAD I were destined to be primary divers for SHAD II, but one withdrew and was replaced. New control subjects were selected from the same pool.

Procedures were worked out for the long SHAD dives to permit a "restart" during the same 30 day period in case one or both divers aborted and had to be replaced. This was to permit utilization of the manpower resources already committed to the project. For a single diver abort at any time a standby diver would replace him as soon as medically feasible. For a two-diver abort or mechanical delay the SHAD profile with revised excursions would continue if 14 days remained, or only the saturation phase if less time. No restart would be considered after Day 23.

After the initial SHAD dives the same screening criteria were used but with a less structured selection process. Some UDT and SEAL divers were used when the available pool of classical divers could not supply subjects. Because local divers were not allowed to draw extra subsistence ("travel pay") there was less incentive for them to participate.

Table II-1 summarizes the characteristics of the SHAD-Nisat divers. Most of the information given there is taken from the records of the Longitudinal Health Study (Sawyer and Baker, 1972; Tansey, 1974; Dembert, et al., in press), which includes data from several hundred USN personnel from which standards are being derived. The LHS records contain a great deal more about the divers than is given in the table.

"Diver number" in Table II-1 is as assigned in Table I-1; the order was supposed to have been alphabetical but this pattern, evidently, was not always followed. There is a question about the "diver number" designation of the Nisat/He III divers and limited documentation about some others; fortunately, all data were recorded by name or initial so no ambiguity exists at that level, and all information in this report is by initials.

Age is given to the nearest birthday at the time of the dive; it changed in divers who participated more than once. Diving experience is as given in the LHS survey. The diver qualification is according to the USN criteria and shows the highest level attained. Divers with UDT and SEAL designation are scuba oriented combat divers and are not necessarily First or Second Class (deep sea) divers. The Saturation divers are also First

Table II-1. Diver Physical Characteristics

	Diver no.	Age, yr	Diving exper, yr	Diver's Naval qual.	Ht, in.	Wt, lb.	Skin fold, mm	FVC, liter	MVV, l/min	Cigs, packs/ day
Pre-SHAD										
GA	1	31	-	-	72	-				Cigars
TT	2	36	-	1st	71	-				0
SHAD I										
WB	1	28	6	1st	71	190	34	5.8	170	1
SW	2	27	6	1st	69	154	25	6.4	203	0.8
PD	c1	25	6	-	67	145	-	6.0	125	-
PP	c2	41	16	Satn	71	185	38	4.7	176	1.5
GS	c3	24	10	1st	69	142	-	5.3	200	0
SHAD II										
GS	1	24	10	1st	69	142	-	5.3	200	0
RF	2	35	12	Satn	69	213	39	4.6	140	0.5
WB	c1	28	7	1st	71	198	34	5.8	170	1
SW	c2	27	6	1st	69	154	25	6.4	203	0.8
TW	c3	-	-	-	-	-	-	-	-	-
SHAD III										
DM	1	37	19	2nd	71	180	31	5.2	251?	0
RO	2	27	4	1st	76	220	-	7.1	202	0
PP	3	42	17	Satn	71	185	38	4.7	176	1.5
Nisat I										
JB	1	33	10	1st	72	226	39	6.6	188	ex 2
RJ	2	34	12	1st	71	223	35	5.6	211	1
NT	3	37	18	Satn	68	161	-	5.5	168	ex 1
Nisat/He II										
JC	1	35	13	1st/UDT	72	215	32	6.8	156	1
MH	2	25	6	UDT/SEAL	69	213	-	5.9	147	0
RO	3	28	6	1st	76	220	-	7.1	202	1
Nisat/He III										
RE	1	25	5	UDT/SEAL	71	165	15	6.1	175	0
TG	2	22	3	1st	73	192	30	7.3	197	0
RL	3	21	3	UDT/SEAL	73	186	24	7.3	223	0

Class. The skin fold thickness is mid-axillary, just above the waist, intended as an additional measure of body build. Pulmonary functions shown include maximum voluntary ventilation and forced vital capacity. Smoking habits are in 20-cigarette packs per day; reformed smokers are designated "ex" and their former level. Missing information was not readily available.

2. Controls

The control subjects were volunteers whose job was to undergo the same medical evaluation as the divers but under normal environmental conditions. This was mainly for the biochemical tests and pulmonary function. Control subjects were monitored during the SHAD I and II dives. As mentioned, the two divers not chosen as subjects for SHAD I acted as controls, and performed topside watch and other duties. One of these (PD) withdrew shortly after the pressure phase started (75 Oct 2, Dive Day 2) and was replaced by another (PP) on Dive Day 10. Both of the control subjects completing SHAD I later served as subject divers, GS in SHAD II, and PP in SHAD III. Controls for SHAD II also changed, with TW working only through the pre-dive period, SW thereafter.

3. Ethics of human experimentation

The SHAD experiments came at the end of a rather long hiatus in human experimental work at the NSMRL. During this period significant changes had taken place in the public's expectations regarding the treatment of experimental subjects. These rules for the ethics of human experimentation were in the process of being formulated by the government (for the first time the requirements for care of human subjects surpassed those of experimental animals!). Because of the sensitivity of this issue it was necessary for approval to be acquired from the Secretary of the Navy before the SHAD experiments could proceed, and this was done.

The procedures followed were quite thorough. A Human Volunteer Consent Committee included members from outside the lab and formally approved by the C.O. It reviewed and approved all protocols. The subjects were thoroughly briefed on the objectives of the experiments, what was to be done, the risks involved and their role as volunteers, and each signed an "informed consent" statement documenting these points. The protocols submitted for clearance were followed explicitly; for example, a test requiring a drop of blood from the earlobe could not be added because only the finger prick had been approved for this type of sample.

In retrospect the procedures developed for handling the ethical aspects of the SHAD and Nisat experiments and fairness to the subjects were at least equivalent to present day practice.

4. Pre- and post-dive procedures

In order to access subtle effects of an environmental exposure it is necessary to control other variables and to determine baseline parameters. Both of these steps were taken in all dives, to varying degrees. Divers were monitored for at least one week, 2 weeks in SHAD, prior to going to pressure.

During the two weeks prior to the SHAD dives the volunteers were encouraged to spend as much time together as possible. Training schedules were varied to accomplish cross training of diver assisted tasks between the various volunteers. Twenty-four hour urine collections and fluid intake logs were maintained during this period. Smoking was not permitted and alcohol intake was limited to two ounces per day. Personal contact between the volunteers and the topside management personnel was maximized to assure familiarization.

The pre-dive period did not follow a vigorous schedule during the SHAD dives, resulting in some compromise of the "purity" of the sampling conditions. For example, a test normally done during a fasting condition ended up being done in mid-afternoon; either the subject had eaten, or he could have been so hungry and hypoglycemic his performance would be affected. This was corrected after SHAD.

After the SHAD dives a two week post dive period was dedicated to medical and physiological evaluations of the divers and a facilities debriefing. After surfacing, the divers were intensely monitored for five days and with a decreasing intensity for the remaining nine days. Alcohol intake was not permitted for five days, and was limited to two ounces per day for the remaining nine days. With a few exceptions this regimen was followed. At the termination of the two week post dive period, the volunteer divers returned to the parent activities. The SHAD subjects were periodically evaluated for an additional period to verify a complete return to normal physiological baselines.

D. Medical management plan

An extensive and quite comprehensive "medical protocol" evolved over the course of the seven SHAD-Nisat dives. It covered all emergencies unique to the situation which could be forecast as being reasonably likely to happen. Except for minor illnesses and the treatment of decompression and counterdiffusion sickness none of these contingency plans was needed.

The plan evolved over a period of 3 years and 6 major experiments. Because it may be of use to others it is outlined here in some detail. A specific plan was not issued from the several drafts prepared for SHAD I, but identifiable plans were issued for SHAD II, SHAD III, Nisat I, and Nisat/He II and III. The outline given here is a composite of these.

The medical plans included many decompression tables to be used under a variety of potentially troublesome situations. These tables were either selected from available ones (such as the U.S. Navy and NOAA Diving Manuals) or were calculated especially for this purpose: The calculated ones were based on the same established NOAA OPS criteria used for calculating the operational tables (see Chapter III). Because they have been given only limited (and inadequately documented) testing we have elected not to include the profiles here, only descriptions.

The first four articles in this section deal with medical preparations, the others deal with specific contingency situations.

Some of the more operational items included in the original medical protocols are covered in the preceding section on safety and emergency procedures. Also, some of the "medical protocol" information related to subject briefing and examination is covered in section E of this chapter.

1. Medical Advisory Committee and Topside Medical Watch

A committee of diving and submarine medical officers at NSMRL was responsible for the medical surveillance of the SHAD I and II divers. Dr. C.A. Harvey was chairman, and the commander of the lab and other qualified medical officers were members, and the Project Coordinator and a dental officer were ex-officio members. Medical specialists were also available by phone.

One of the medical officers on the Committee locked into the chamber each day and completed a physical examination on the divers. The committee convened each morning of the dive to review the physical exam findings and the serial biomedical monitoring data. Based on this information a decision was made each day to continue or terminate the dive.

A qualified Diving Medical Officer, one of the group, was immediately available in the building at all times. He remained at the chamber during changes in pressure or in the chamber atmosphere.

After the two SHAD's, the structure of the committee was changed and daily physical examinations were no longer conducted by "lock in." The Topside Medical Watch was composed of the duty Diving Medical Officer (who was responsible for the health of the divers and for advising the project director on medical matters), a Diving Corpsman (who implemented matters of "patient care" as well as standing chamber watch), the Diving Supervisor (who was responsible for operational matters relating to health), and the standby Diving Medical Officer (who acted as a consultant to the duty Medical Officer). Each day the duty Medical Officer interviewed the divers by phone and through the port, and reviewed the serial biomedical monitoring data. The other duty Medical Officers or specialists were consulted if there were any questionable findings.

One diver in each team was trained as a medical corpsman.

2. Diver physical examinations

The SHAD divers (and controls) had all been medically qualified for diving previously and were all examined in detail for the Longitudinal Health Study (Sawyer and Baker, 1972; Tansey, 1974) some 2 weeks before the dive. LHS is a multi-phasic health screen consisting of a complete family and personal history, a PA and lateral chest film, pulmonary function tests (both static and dynamic lung volumes), anthropometric measurements, dental exam, audiogram, visual testing including fundus photos, orthorator, color vision, tonometry and refraction, EKG, CBC, urinalysis, VDRL, SMA-12, serum banking for future studies deemed desirable, and the Minnesota Multiphasic Personality Inventory.

A recheck was given about 2 days before and immediately before going to pressure. Special attention was given to avoiding contagious diseases. Each diver received conservative preventive dental treatment before the dive. Results of the physicals were studied thoroughly by the watch Medical Officers to serve as a baseline and help them interpret their daily observations.

During SHAD I and II brief daily examinations were carried out. The Log Recorder topside filled out a form and thus ensured that no items would be missed. The daily exam stressed subjective complaints or symptoms of the divers, the usual vital signs and reflexes, eye, ear, mouth and skin inspection, and nerve and sensory function.

In addition to this medical surveillance the project leader interviewed each diver each day, usually privately.

In keeping with experimental ethics, if a diver at any time were to decide he could not continue for emotional or other reasons, he was to make this known to the duty medical officer. Every effort would then be made to allow him to decompress and withdraw as soon as safety allowed.

3. Serial biomedical data

The biomedical monitoring which comprised a significant part of the experimental aspect of the SHAD-Nisat program was also important in its medical management.

A Data Coordinator was specified for each dive. His duty was to see that the raw results of all available data were plotted and displayed. Tables and hand-drawn graphs were posted in the chamber room and kept up to date with analytical results as they came in. This gave a serial view of these hundred or so parameters on each diver. This information was invaluable to the medical officers in making their daily assessments.

As a minimum the serial data display was to reflect daily progress of vital signs (heart rate, respiration rate, blood pressure), body weight, chamber atmospheric parameters, blood counts, urinalysis, EKG, pulmonary function and Doppler bubble detection.

4. The Medical Diving Locker

A cabinet of medical supplies was kept handy to the chamber. It was maintained by the Diving Corpsman and inspected by the Medical Department before the dive. Its contents are given in Appendix B.

5. Compression phase

For compression with air the primary complications are squeeze and barotrauma. The pre-dive exam should evaluate the diver's ability to clear his ear and sinuses. The compression rate (60 fsw/min) is not critical for this type of dive, and compression was stopped, slowed or reversed momentarily if a block occurred; divers with ear block problems were given Sudafed or Auralgan decongestants. The symbol to stop compressing is to raise a clenched fist.

Headsets or ear defenders should be used by the chamber occupants during rapid compression for protection against the noise.

When compression was with inert gas divers breathed air by mask during descent. One diver removed his mask first, the second 5 minutes later and the third in turn. The divers were observed by topside and each other; if there had been anomalies other than the predictable narcosis the mask removal would have been stopped and the Medical Officer would decide whether to continue or go back on mask.

If abort had been necessary at this point the divers would breathe air by mask until the chamber could be purged with air.

6. Saturation depth exposure

Limits for the saturation environment were predetermined, as follows.

Oxygen: For SHAD, limits were 19-21.5% oxygen, optimum 20.9%. For Nisat I the target was "normoxic" or a PO_2 of 0.21 atm; this was later changed to 0.3 atm, and this level was used in the later Nisat dives.

Carbon dioxide: Original maximum was 0.2% surface equivalent (or a PCO_2 of 2 mmHg), later this became a target value and 0.5% SLE was the maximum (4 mmHg).

Humidity: 65 to 80% was selected for SHAD I but this was later changed to 45-80%; the criterion here is diver comfort, but high humidity tends to promote ear infection.

Temperature: Range 70°-85°F, to be comfortable for divers.

Carbon monoxide: Maximum of 20 parts per million.

Hydrocarbons: Maximum of 10 mg/m^3 (as methane).

Hydrogen sulfide: Maximum of 20 ppm.

The same type of precautions mentioned above regarding compression with inert gas were followed during the isobaric switch. The divers made the transition in turn, during which time their electrocardiograms and well being were carefully monitored.

Particular care had to be paid to preventing external ear infection. Prophylactic ear medication was not employed in SHAD, but the Nisat divers were required to instill Domeboro Otic solution 3 times daily at mealtime and after showers. These treatments were timed (5 min each ear) by topside.

Food sanitation was monitored daily by the person responsible for food procurement.

7. CNS oxygen toxicity

There are no completely dependable subjective or objective warnings to indicate that an oxygen convulsion is about to occur. However, the divers and crew should be alert for the following findings, particularly during the excursions: Constriction of the visual fields (tunnel vision), retinal vasoconstriction, other visual abnormalities, changes in hearing, incoordination, twitching, numbness, tingling, dizziness, and confusion.

If a strong symptom occurs the diver's exposure to high oxygen should if possible be terminated. If on mask, remove the mask; if the hyperoxic exposure is from the chamber atmosphere, then the diver should breathe a lower oxygen mixture by mask (in SHAD-Nisat, 90-10 N_2-O_2). A convulsion may occur even after hyperoxic exposure has been terminated.

If a convulsion does occur the diver's oxygen level should be lowered and he should be gently restrained to prevent injuring himself during the vigorous and generalized clonic contractions. Excessive restraint should be avoided. During the initial tonic phase at the onset of the convulsion, the head becomes hyperextended and the lower jaw is strongly depressed so that the jaws are separated. During this period of about 10 seconds, a soft but firm "bite lock" such as a padded tongue depressor can be inserted to prevent chewing of the tongue during the subsequent clonic jaw clamping.

During both the tonic and clonic phases the diver is unconscious and there may be interference with pulmonary ventilation. It is therefore extremely important to avoid decompressing during any part of the convulsion, to prevent embolism.

Within a few minutes after the convulsion the diver will begin to breathe again. At this point it is especially important to maintain an open airway; the diver may have soft tissue obstruction and a accumulation of unswallowed and excessive salivary excretions. In a few minutes, after breathing is regular, it is safe to begin decompression. During the return of consciousness the patient may be irrational and will at least require reassurance and possibly gentle restraint to prevent confused activity and self injury. At times consciousness returns abruptly and the patient shows

surprising mental clarity. Headache or nausea may occur, and muscular fatigue is to be expected. Consciousness and normal central nervous system function should return within a few minutes to an hour after the convulsion. Following a convulsion, severe headache may result. This can be treated with aspirin, rest and sleep.

In SHAD I it was planned that a diver having a convulsion during an excursion could be decompressed using a special schedule to 50 fsw, be stabilized there, then be transferred to the standby chamber for subsequent decompression. Special schedules were available to permit the decompression from an excursion in case the diver had to remain there longer than planned and breathe 90-10 N_2-O_2 mix.

8. Pulmonary oxygen toxicity

During the type of exposures involved in SHAD (Nisat did not involve hyperoxic exposure except during treatment) it is possible that some small changes in vital capacity of the lungs will occur. The medical staff should also recall that the following signs, symptoms and laboratory results have been reported during pulmonary oxygen toxicity: Swelling of nasal mucous membranes, progressive respiratory insufficiency, dyspnea (particularly on exertion), substernal discomfort, cough, congestion, labored breathing, apnea, rales, ronchi, bronchial breath sounds, copious tracheal secretions, atelectasis, pulmonary edema, asphyxia, frothy or bloody sputum, tracheo-bronchitis, pneumonitis, cyanosis and shallow breathing. Also reported are fever, weakness, anorexia, nausea, vomiting, undue restlessness, lethargy, decreased total lung capacity, decreased diffusing capacity, and decreased compliance. Radiologically one sees diffuse, bilateral pulmonary reticulogranular densities which can extend and coalesce. The main symptoms are chest pain and coughing.

The treatment philosophy is based on the fact that these changes can be expected to occur gradually, and that removal of the diver from the hyperoxic environment will terminate the toxic symptoms and allow recovery to begin immediately. A mixture of 90-10 N_2-O_2 can be used deeper than 50 fsw (i.e., PO_2 of 0.5 atm) to give the affected diver some relief. In a case where development of symptoms is excessive the diver should be given symptomatic, supportive treatment after removing the oxygen stress.

In SHAD the procedure called for putting the diver on 90-10 N_2-O_2, decompressing him to 50 fsw and there having him breathe 90-10 N_2-O_2 by mask; he could remain there for as long as desired before decompression on a special schedule calculated for the anticipated exposure that used 90-10 and 85-15 N_2-O_2. If appropriate, procedures were also developed so that he could be transferred to the standby chamber and decompressed there while another subject took his place.

9. Saturation decompression

This part of the original medical protocols is covered in Chapter VI.

10. Treatment of DCS (Decompression sickness)

The management of decompression sickness in SHAD involved special consideration for excursions and for divers who might have pulmonary oxygen toxicity. Nisat involved an especially deep nitrox exposure and the isobaric switch of inert gas. Where possible existing procedures were chosen, but for SHAD special treatment profiles were computed. Computation methods used are covered in Chapter VI.

a. DCS after excursions

During SHAD I a diver with DCS following a descending excursion was to be given 2 cycles of oxygen at 50 fsw (a "cycle" here was 20 minutes on oxygen, 10 minutes on air), then if not relieved was to be recompressed. Recompression of this diver or one with DCS following a descending excursion was to be to one of the depths for which a special schedule had been prepared. These schedules allow the use of 90-10 N_2-O_2 for relief of pulmonary oxygen stress.

In SHAD II recompression after the 2 cycles was to be in 10 fsw increments (at 60 fsw/min) to depth of relief, holding 3 minutes at each stop, until relief. A contingency table for return to the habitat was to be calculated at the time (or one from SHAD I would be used).

In SHAD III recompression was to be at 30 fsw/min in 10 fsw increments, holding 2 min at each stop. This recompression was not to exceed 130 fsw; a special table for decompression from 130 fsw was on hand which allowed 90-10 nitrox to be breathed while deeper than 50 fsw.

b. CDS after inert gas switch

Symptoms of counterdiffusion sickness (CDS) in Nisat/He II and III were not expected, but plans were made to deal with it; significant symptoms did occur and treatment was used. Predominant CDS symptoms would be itching, often intense, with blisters and blebs forming on exposed skin. Symptoms of serious CDS are the same as serious DCS and include dizziness and vertigo, change in vision and hearing, weakness, tingling, chest pain, shortness of breath, speech difficulty, tremors, and other symptoms or signs resembling embolism.

Treatment would only be needed after the switch to helium. Procedures called for recompression in 10 fsw steps to depth of relief, as deep as 165 fsw if symptoms are serious, and breathing of 4 cycles (20 on, 5 off) of treatment mix providing a PO_2 of 1.5 to 2.5 atm. Decompression was to be in heliox if possible or nitrox if necessary, at 0.3 atm PO_2.

The nature of the recompression and background gases were not clearly stated in the plan, but were left up to the Medical Officer and Supervisor.

c. DCS during saturation

For mild symptoms 2 cycles (in this case 20 min O_2, 10 min air, 20 min O_2) at onset depth should be tried. If symptoms did not clear up SHAD plans called for recompression at 30 fsw/min in 10 fsw stops holding 10 minutes at each stop. It should not be helpful to recompress beyond 60 fsw or to 30 fsw deeper than onset. Oxygen cycles (20-5) were then to be breathed until 2 cycles past relief. Decompression would be by the "conservative" (or standard air) NOAA profile (28 1/2 hours from 60 fsw).

For SHAD III the procedure was equivalent to that given for DCS following excursions, above. The procedure actually used is given in VI.C.

For Nisat I recompression up to 30 fsw at 0.5 fsw/min was to be used, with 6 cycles of oxygen or treatment mix at relief depth. The medical officer was then to prepare the post-treatment schedule.

Nisat/He II and III involved the standard USN heliox decompression schedule and the appropriate treatment was to be followed if needed. It would be an understatement to note that these treatment procedures have been well tested.

11. Miscellaneous problems

Several more or less remote contingencies were considered and covered by the plan.

a. Evacuation of chamber or transfer of a diver

In the event that the Genesis chamber (#1, Fig. 2-1) were to become unuseable or in case a single diver had to be removed during an ongoing experiment, plans were made to use a backup chamber (#2) for the saturation decompression. For the saturation depths used in SHAD a 10-minute surface interval had been shown to be safe. Ascent was to be as fast as possible, the surface interval not over 10 minutes, and recompression at 60 fsw/min back to saturation depth (or to treatment depth if necessary).

For SHAD I and II the plan was to transfer to the chamber at the submarine escape training tower elsewhere on the submarine base. This transfer was drilled, and it was demonstrated that it could routinely be accomplished in 9 minutes, with one trial as short as 7 1/2 minutes. After SHAD II a scrubber was installed in Chamber 2 making that chamber useable, and transfer could then be accomplished in a matter of seconds.

b. Contaminated or hypoxic atmosphere

Divers would breathe by mask, air normally or an appropriate mix, and action would be taken based on the circumstances.

c. Sudden decompression

If a porthole, hatch seal or penetration were to fail the chamber would undergo a rapid or "explosive" decompression. The first step, hardly necessary, is for divers to "notify topside." During the decompression they are then to perform the "ho, ho, ho," manuever, a series of yells or purposeful exhalation to ensure an open airway. They then proceed to the other lock, secure the hatch and are repressurized with air. If necessary they can be recompressed in the standby chamber.

The only case where this eventuality posed a significant risk (except for the everpresent possibility of embolism) was in Nisat I, where the PO_2 was 4% (3% for a while!) and would create a hypoxic environment which would be a far greater threat than even embolism. It was not stated explicitly in the protocol, but topside's first action in such a case should be to start a full purge of the chamber with air. This they would do in an attempt to maintain pressure anyway. The gas used should be air rather than nitrogen or a low oxygen mix. The first thing the divers should do in this special case is escape if possible, if not they should go on mask. It is imperative that the crew and divers all understand that hypoxia is the real hazard.

This is also an argument favoring leaving the outer lock at depth rather than at the surface. (Fire risk also supports this option.)

d. Electric shock

All strong electrical leaks are to be reported. If a diver is shocked, other divers notify topside and initiate resuscitation if the diver is not breathing, and if necessary the medical officer locks in with a defibrillator.

E. Biomedical monitoring

1. Specialized laboratories

The NSMRL is particularly well equipped to perform comprehensive biomedical monitoring of human environmental exposures. The several departments have a number of specialized laboratories, each of which can bring to bear its technical expertise and instrumentation on a specific aspect of the subject's response. The departments participating in SHAD-Nisat were Physiology, Biochemistry, Auditory, Vision, Human Factors, Psychology, Ecology, and Oral biology. The experiments themselves were run by Military Applications, later to become the Environmental Medicine Department.

The Project Coordinator or Principal Investigator organized the timing of testing and sampling, and as much as possible eliminated redundant sampling and endeavored to prevent areas of coverage from being forgotten.

2. Parameters monitored

During the course of the project over 100 biomedical parameters were examined. The items for which samples were taken and/or measurements made are listed here, but in some cases nothing more is heard from regarding the parameter. Most data taken were analyzed, but for several reasons not all of these led to completed results. In some cases the data did not show differences that could be attributed to any known aspects of the environmental exposure. In the case of a few parameters the methodology could not be made to work satisfactorily and the monitoring had to be abandoned. In other cases control information was inadequate and/or the scatter too great to make further interpretation worthwhile. All cases where significant changes were noted are reported, either in the publications listed in section IX.A or in this report.

Not all parameters were followed in all dives. For example, those tending to reflect the development of oxygen toxicity were given less emphasis in the nearly normoxic Nisat dives, and some showing no changes in the early SHAD measurements were not followed later.

Biomedical measurements made at one time or another during SHAD-Nisat include the following. Specific methods are given with results, if appropriate.

a. Biochemistry

(1) Serum chemistry

Creatinine
Uric acid
Glucose
Lactose
Iron
Serum osmolarity
Angiotensin I
Aldosterone
Antidiuretic hormone
Blood urea nitrogen
Bilirubin, total
Phosphate, inorganic

(2) Serum electrolytes

K^+, Na^+, Ca^{++}, total Ca, Mg^{++}, Cl^-
Phosphate, inorganic

(3) Serum lipids

Cholesterol
Total lipids
Triglycerides

(4) Serum proteins

Total protein
Albumin
Alpha-1 globulin
Alpha-2 macroglobulin
Beta globulin, pre-beta
Gamma globulin
T_3, T_4, T_7 (free thyroxine index)
Haptoglobin
IgA
Complements C'3, C'4, C'5
1-Antitrypsin
Alpha-1-acid-glycoprotein
Alpha-2-glycoprotein
Beta and gamma glycoprotein

(5) Serum enzymes

LDH
SGOT
SGPT
CPK
Alkaline phosphatase

(6) Blood gases

Blood pH
P_aCO_2
"Actual" bicarbonate; base excess
P_aO_2
$\%_a$ oxygen saturation
Carboxyhemoglobin

b. Urinalysis

Volume (each void and 24-hr total)
Osmolarity
Titratable acidity
Electrolytes
Na+, K+, Ca++, Cl-, Mg++
Phosphate
Sugar
Creatinine
Blood
Cells
Ketones
Protein
Hydroxyproline
17-ketosteroids
17-hydroxysteroids
Urea
Uric acid

c. Hematology

Red blood cell count
Hemoglobin
Hematocrit
Red cell
  Na+, K+, Cl-, Ca, phosphates, volumes, water
Platelet count
Differential WBC counts
Reticulocytes
Polymorphonuclear lymphocytes
Eosinophils
Neutrophils
Lymphocytes
MCH
MCHC

d. Pulmonary/respiratory function

Respiratory frequency
Inspiratory/expiratory time ratio
FVC, including, FEV_1, FEV_2
MIFR, MEFR, MVV
Closing volume, as fraction of FVC
Inspired-expired gas analysis (O_2, CO_2, N_2)
Nitrogen gas exchange
CO_2 respiratory response
Exercise tolerance

e. Vital signs and physiology

Rectal temperature
Body weight
Food/waste balance
Fluid balance
Blood pressure
Heart rate
EKG, 12-lead scalar with derivatives
Vector EKG, pre and post
Doppler bubbles
Long-bone skeletal x-ray, pre and post
Bone density, pre and post
Longitudinal Health Survey exam, pre and post

f. Psychological assessment

MMPI
Depression proneness sentence stems
Diver biographical inventory
Interview, daily and/or pre and post

g. Human factors

Adaptive tracking
Mental arithmetic
Short-term memory
Sentence comprehensive
Signal detection
Time estimation
Letter cancellation
Ball bearing psychomotor test
Nut and bolt test (Bennett Hand Tool Dexterity)
Visual reaction time, bright and dim

h. Audiometry

Audiogram, pre and post
ENG, pre

i. Vision

Visual aciuty
    Far and near, left and right, binocular
Phorias
    lateral and vertical, far and near
Depth of vision
Field of view
Color vision (FM 100-hue test)
Night vision
Fundus photography
    Artery and vein diameter, A/V ratio
    Bubble surveillance

j. Brain function

(1) EEG
    Alpha and theta, eyes closed and open
    Beta

(2) Visual evoked responses (VER)
    Striped and blank fields
    1 and 16 flashes/sec

k. Oral biology

Dental exam, pre and post
Intra-oral photography, pre and post
Mouth plaster cast, pre and post
Salivary bacteria count
Parotid fluid
    Flow rate
    Adrenocorticosteriods
    pH
    Electrolytes
        Na^+, Cl^-, K^+

1. Microbiology/Ecology

Bacterial counts
- Skin, oral, nasal
- Airborne
- Potable water
- Mechanical environment

F. Order of events

1. Overall dive schedule

Table I-1, page I-7, summarizes the complete SHAD/Nisat series, over a 3-year period lasting from September 1973 to July 1976. The overall monitoring period is shown for each dive ("Exptl period") as well as the time when the divers were actually under pressure. The dives are displayed graphically in the frontispiece.

2. Dive plans

Each of these experiments involved repeated evaluation of relevant aspects of the physiology, biochemistry, performance and medical status of the diver-subjects, for periods of up to 2 months. These were conducted by several groups of investigators. Their observations were (or could have been) affected by many factors, including the stage of the "dive," the time of day and the diver's other activities, including the tests themselves. It would have been impossible to integrate all these activities without a detailed schedule of events. Accordingly, detailed protocols covering all active dive days and many pre-and post-dive days were prepared before each operation.

These are voluminous and impossible to include here. A good part of what actually happened is in the logs, and frequency of sampling is covered in some experimental protocols.

The vital signs were measured daily by the divers themselves. The radial pulse and respiratory rate were obtained by the usual clinical method of timed palpation and observation. A sphygmomanometer was used to record the arterial blood pressure, either a conventional aneroid or a solid state B650 Sphygmostat (Technical Resources, Inc., Waltham, Massachusetts). The rectal temperature was recorded by a rectal probe thermister (Model 4002 Switch Box, plus YSI Model 42SF Tele-Thermometer, Yellow Springs Instruments, Ohio). Body weights were obtained using a commercial household bathroom spring scale throughout the study. During pre- and post-dive the vital signs were measured when the divers arrived at the Laboratory in the morning; when under pressure, they were obtained after reveille and before breakfast.

3. Representative daily protocols

To provide a picture of the organization of a day's activities this section presents some typical daily protocols. These are essentially the same as those used for carrying out the dive operation, but some lock transfers, etc., have been omitted (p. 37-39).

Pre-SHAD did not use a prepared protocol. SHAD I and SHAD II followed almost the same pattern (except for the difference in residence depth) so a single protocol, Table II-2, shows a "typical" day for both of those; this is Dive Day 12 of SHAD I.

Shad I & II had a main schedule prepared and portions of a schedule which were interchanged according to the daily excursion to be performed. All non-excursion days have the same schedule and all days with an excursion at 1000 hr have the same schedule, etc.

Because of the symmetry of SHAD III it was possible to use the same daily schedule for each excursion day, with the alternation of some tests. Dive Day 3 from SHAD III is given in Table II-3. Nisat I also had a relatively consistent schedule, shown in Table II-4.

Nisat/He II and III were to follow the same schedule, only at different depths. The SHAD I schedule for Day 4, the day of the switch, is given in Table II-5. The other days resembled those of Nisat I.

G. The experimental environments: What was done

The actual environments to which the divers were exposed are covered in several places. The time lines and the logs show the day to day environmental conditions; special aspects of a given experiment are covered with the experiments.

1. Time lines

Day by day time lines for all dives except Pre-SHAD are given here, in Figures 2-2 through 2-7 (p. II-40 ff.). These show the pressure profile of the dives in simulated feet of sea water, the partial pressure of oxygen in atmospheres, the Fahrenheit temperature, and percent relative humidity. The latter parameter is unreliable in the Nisat dives, and in Pre-SHAD. Also, the diver's body weights (pounds) and rectal temperature (°F) are displayed on the same time scale. Carbon dioxide levels were kept below the level of physiological significance so are not displayed.

2. Operational logs

A log was kept during all times that divers were in the chamber, hand written by a person whose primary shift duty was as the recorder. These logs were recorded in the format set up by the International Decompression

Data Bank at the Institute for Environmental Medicine, University of Pennsylvania (Bardin, 1973), and copies were submitted for inclusion therein.

Information recorded included the following items: Date; either time of day or running time of the dive; a code to identify the item being logged, e.g., a chamber, person, test or remark; the identity of the coded item, such as the chamber or person's initials; an action entry showing if the event is "at" a given place or depth, or leaving or arriving; the place or depth; a further action statement; environmental measurements of nitrogen, oxygen, temperature and relative humidity; and remarks. This format has been devised to facilitate analysis of the logs by computer. Remarks included symptoms, medications experimental testing performed by or on the divers, symptoms, lock transfers, meals, visits by others, etc.

Condensed versions of the operational dive logs are given in Appendix A, including a description of the details of how the extracts were made. Briefly, they cover the divers' environment: time, depth, oxygen partial pressure, temperature and humidity. Remarks cover pressure, oxygen, and other significant aspects of the divers' environment, including exercise periods. Housekeeping details are omitted, as are experimental activities, but all symptoms or signs reflecting the divers' condition are included. All Doppler bubble detection readings in the original logs are included, and the profile of all excursions is given. This format was used both to save space in the logs and to permit computer analysis for determination of oxygen dosage. An additional log dealing with oxygen dose is given in section VII.B.

3. Other data

A statistical analysis was run on the daily values during SHAD I. This showed about 45 measurements per day for oxygen, CO_2 and temperature, about 8-10 for humdity. The oxygen partial pressure in the chamber ranged around 0.50 to 0.51+ atm, with a standard error of the mean in the range 0.002 to 0.010. This reflects a well controlled environment. Temperature averaged 76.5° to 78° with a typical SEM of about 1.7. Carbon dioxide, measured 45 or so times daily ranged typically between 0.02 to 0.06 percent (measured at sea level) with an SEM of about 0.002. This produced PCO_2 values in the range 0.4 to 1.2 mmHg. The mean daily SHAD I relative humidity ranged between 59 and 65%.

Sound levels were measured in the Genesis chamber when it was in the configuration for SHAD-Nisat (date uncertain). Ambient levels with everything running were 60 dBA. During compression at 70 fsw/min recordings were as high as 120 dBA; during ascent at 33 fsw/min it reached 115 dBA. These are the classical wide band ("A scale") measures relative to a sound pressure of 0.0002 dynes/cu^2.

Hearing protection, ear defenders or headsets, were used during compression. This was considered optional early in the program; later it was required.

Table II-2
Representative SHAD I & II daily protocol:
SHAD I, Dive Day 12, 73 Oct 12

0600 Dental branch obtain air sample #1.
Medical lock on surface, load whole saliva samplers, rectal temperature probes, coffee (black).
Outer lock load potable water, ice chest, toilet, microbiology rectal swabs, urine bottles, clean clothes.
0610 Dental branch obtain air sample #2.
0615 Medical lock leaves surface.
0620 Divers wake up, start whole saliva samples, obtain inspired/expired gas analysis, weight, rectal temperature, as time permits. Outer lock leaves surface.
0650 Divers secure whole saliva samples, place in medical lock, notify topside. Medical lock travel at 8 ft/min. Divers' field day inner lock (clean up).
0740 Outer lock secured, divers in inner lock. Outer lock leave bottom, travel at 30 ft/min.
0746 Outer lock reach surface, load blood sampling gear, physical examination set up, microbiology sampling gear, diving medical officer, human factors tracking gear, breakfast.
0750 Outer lock leave surface. DMO obtains blood samples. Breakfast.
0830 Diver #1, physical examination. Diver #2, pulmonary function.
0840 Diver #1, pulmonary function. Diver #2, physical examination.
0850 Divers' field day inner lock into outer lock. DMO in outer lock with physical examination set up.
0855 Outer lock leaves bottom, travel at 30 ft/min. Divers attach EKG, VER leads.
0915 Diver #1, human factors I evaluation (tracking, mental arith.).
Diver #2, VER, do not detach electrodes.
0930 Medical supervisory committee meeting (topside).
Diver #2, human factors I evaluation (tracking, mental arith.).
Diver #1, VER, do not detach electrodes.
0945 Both divers, inspired/expired gas analysis.
0950 Topside/divers carry out pre-excursion procedures:
Topside - 90/10 nitrox on line, open dump valves.
Divers - Open dump valves, test overboard dump masks, 10 sec max.
Topside and divers - Long count audio system, channels C & D.
Divers - Open ice chest, open potable water container, set up toilet making sure caps are slightly loosened, check urine bottles, pull plug on blood gas apparatus. Plug in Doppler transducer, apply sonic jelly, sit on lower rack.
1000 Divers brief with project coordinator.
1010 Begin excursion: Inner lock leave 50 feet, travel at 60 ft/min to 235 feet.
Diver #1 in human factors chair, start task when tracking light appears, audio on channel D.
Diver #2 in VER chair with electrodes hooked in, audio channel C.
1013:06 Inner lock reach 235 feet.
1016 Diver #1, switch to VER, commence VER.
Diver #2, switch to human factors chair for tracking.
1019 Inner lock leave 235 feet, travel at 30 ft/min.
Diver #1, position Doppler transducer.
Diver #2, sit on lower rack, stand by for Doppler.
1021 Diver #2, position Doppler.
1023 Diver #1, position Doppler.
1025:07 Inner lock reach 50 feet.
Diver #2, position Doppler for 20 min monitoring.
1029 Divers inspired/expired gas analysis.
1035 Diver #1, position Doppler for 20 min monitoring.
Diver #2, human factors I (tracking, mental arithmetic only).
1045 Diver #1, human factors I (tracking, mental arithmetic only).
Diver #2, position Doppler for 20 min monitoring.
1055 Divers inspired/expired gas analysis.
1100 Diver #1, pulmonary function evaluation.
Diver #2, Doppler monitoring.
1110 Diver #1, Doppler monitoring.
Diver #2, pulmonary function evaluation.
1120 Medical lock leave surface with nasal and parotid fluid samplers.
1125 Divers start parotid fluid collection for 20 min, perform Doppler monitoring, obtain nasal samples.
1145 Divers secure parotid fluid samplers.
1150 Medical lock leave bottom with parotid samples, microbiology samples, travel at 8 ft/min.
1200 Lunch.
1300 Outer lock leave surface with orthorator, bicycle.
1305 Outer lock reach bottom. Divers exercise at 100 watts for 15 min any time after 1400.
1615 Divers set up orthorator, notify topside when power required.
1630 Start orthorator task.
1700 Secure orthorator task, topside secure power.
1705 Diver #1, Pulmonary function. Diver #2, Rectal temperature.
1710 Diver #2, EKG.
1715 Diver #2, Pulmonary function. Diver #1, Rectal temperature.
1720 Diver #1, EKG.
1725 Medical lock leave surface with supper, chamber microbiology swipes. Supper. Divers free time.
Prior to lights out, field day inner lock, place ice chest in outer lock, prepare toilet for surfacing. Bring outer lock to surface prior to lights out. Topside make up O_2, shift CO_2 cannisters if required.
2300 Lights out.

Table II-3
Representative SHAD III daily protocol: Dive Day 3, 74 Dec 5

0600 Reveille - start coffee

Topside -
(1) Loosen caps on all containers to be loaded (except small red top tube in blood draw tubes)
(2) Load medical lock with towels, scale, rectal temp probes, parotid samplers and black coffee. Fresh clothes also via medical lock.
(3) Go over today's schedule with the divers.

Divers -
(1) Psychological log
(2) Parotid samples prior to anything by mouth. Place in medical lock and send to surface.
(3) Weight and temperature. Diver subjective index.
(4) Wash, shave, shower, housekeeping, dirty clothes into outer lock.
(5) Secure extra bunk. Prepare for blood draw.

0715 Outer lock to surface for medical teams, toilet emptying, blood draw equipment, urine collection bottles.

0730 Compress outer lock and medical lock.

0735 Blood draw when ready. When medical samples are vented and ready, place in medical lock and surface medical lock at 8 ft/min.

0745 When medical lock topside notify biochemistry, remove needle vents from blood samples.
Diver #1, assist. #2, physical exam. #3, pulmonary function.

0800 Diver #1, pulmonary function. #2, assist. #3, physical exam.

0815 Diver #1, physical exam. #2, pulmonary function. #3, breakfast.

0830 Diver #1 (DM) and #2 (RO), breakfast. #3 (PP) human factors.

0900 Outer lock to surface between human factors tests, with breakfast dishes, trash, vented urine samples and medical team.

0930 Topside load remainder of equipment needed for day in outer lock. Bacteriologic samples to be obtained today.

0945 Outer lock to 50 feet.

1000 Begin excursion. Diver #3 commence tracking and both locks travel to 100 fsw.

1004 Diver #3, complete tracking. #2 weight lifting, 20 min.

1010 Diver #1, pulmonary gas exchange. #3 assist.

1030 #2, assist. #1, human factors (45 min). #3 gas exchange (20).

1050 #1, human factors. #2, pulmonary gas exchange. #3, assist.

1110 #2, rig EKG. #3, assist.

1120 #1, EKG. #2, assist EKG. #3, rig bicycle.

1130 #1, bicycle exercise, 30 min. #2, assist. #3, EKG.

1140 #2, forced vital capacity. #3, weight lifting (20 min).

1200 Lunch.

1250 #1, forced vital capacity. #2, EKG.

1300 #3 exercise tolerance. #3 forced vital capacity. Weight lifting Diver #2 (20 min).

1330 #2, human factors (45). #3, bicycle exercise (30).

1415 All divers, audiogram and EEG/VER.

1600 Lock in Medical Team with blood gas equipment. Set up equipment, purge with nitrogen, blood draw when ready.
Parotid fluid collection, all three divers, Diver #2 first.

1640 Exchange blood gas technicians and lock out physician.
Pulmonary function, Diver #1 (DM), then Diver #3 (PP).

1720 Blood gas technician to outer lock and to surface. #2 pulmonary function.

1745 Prepare for Doppler monitoring.

1800 Begin decompression to 50 fsw during a twenty minute period at two and one-half feet per minute (1 foot every 24 seconds). Doppler monitoring of 3 divers alternately.

1820 Audiograms, all 3 alternately.

1820 Continue Doppler monitoring intermittently for next hour.

1920 Supper.

2000 Movie.

2130 All 3, Electrocardiograms.

2200 All 3, forced vital capacity.

2300 Psychological status logs; taps.

Table II-4
Representative Nisat I daily protocol
Dive Day 5, 74 Mar 16

0630	Reveille. Complete objective check list, gas exchange, vital signs.
0640	Diver #1, gas exchange. #2, rectal temperature. #3, weight.
	Diver #1, blood pressure. #2, respiration. #3, pulse.
	All, blood draw and personal cleanup.
0800	#1, pulmonary function. #2, balance beam. #3, cancellation.
	All, breakfast.
0900	Diver #1, tracking. #2, short term memory. #3, visual reaction time.
	Diver #1 and #2, mental arithmetic.
	Diver #1, reaction time. Diver #2, nuts and bolts.
	Diver #1, visual reaction time. #2, tracking. #3, short term memory.
	Divers #2 and #3, mental arithmetic.
	Diver #2, reaction time. Diver #3, nuts and bolts.
	Diver #1, short term memory. Diver #2, visual reaction time. Diver #3, tracking.
	Divers #1 and #2, mental arithmetic.
	Diver #1, nuts and bolts. Diver #3, reaction time.
	Diver #1, gas exchange. #2, assist. #3, digit span.
	Diver #1, digit span. #2, gas exchange. #3, assist.
	Diver #1, assist. #2, digit span. #3, gas exchange.
1200	All, lunch.
1315	Diver #1, EKG. Diver #2, MD interview. Diver #3, audiogram.
1324	Diver #1, audiogram. Diver #2, EKG. Diver #3, interview.
1344	Diver #1, interview. Diver #2, audiogram. Diver #3, EKG.
1445	Diver #1, color vision. #2, assist. #3, exercise tolerance.
1526	Diver #1, exercise tolerance. #2, color vision. #3, assist.
1544	Diver #1, Assist. #2, exercise tolerance. #3, color vision.
1700	Diver #1, tracking. Diver #3, short term memory.
	Diver #1 and #3, mental arithmetic.
	Diver #1, reaction time. #2, time estimation. #3, nuts and bolts.
	Diver #2, short term memory. Diver #3, tracking.
	Divers #2 and #3, mental arithmetic.
	Diver #1, time estimation. #2, nuts and bolts. #3, reaction time.
	Diver #1, short term memory. Diver #2, tracking.
	Diver #1, mental arithmetic. Diver #2, nuts and bolts.
	Diver #1, nuts and bolts. Diver #2, reaction time. Diver #3, time estimation.
1800	All, dinner.
1900	Diver #1, stand-by. #2, cancellation. #3, balance beam.
	Diver #1, balance beam. #2, stand-by. #3, cancellation.
	Diver #1, cancellation. #2, balance beam. #3, standby.
2030	All, free time.
2200	Diver #1, ball bearing. #2, time estimation. #3 reaction time.
2215	All, Complete adjective check list.
	Taps.

Table II-5
Representative Nisat/He II & III daily protocol
Nisat/He II, Dive Day 4, 76 Jun 11

0600	Reveille.
	All divers, body weight, respiration rate, temperature, 12-lead EKG, blood pressure.
	All divers, personal cleanup.
0720	All divers, blood draw.
0730	All divers, breakfast.
0800	All divers, parotid study.
0815	All divers, set up body plethysmograph, fundus camera, and check related systems.
0900	All divers to outer lock, begin placement of electrodes for EKG, VER and Doppler. Divers #2 and #3 assist #1.
1100	Diver #1, enter inner lock, put on headphone, connect leads, begin monitoring.
1105	Diver #1, set up plethysmograph box, flush bags.
1120	Diver #1, continue monitoring Doppler, EKG and EEG.
1130	Diver #1, VER and EEG. Divers #2 and #3, lunch.
1140	Diver #1, Doppler, EKG and EEG.
1150	Diver #1, 12-lead EKG.
1205	Diver #1, Doppler and EKG.
1230	Diver #1, get things ready for #2.
1300	Diver #1, assist #2. Diver #2, enter inner lock, breathe in bag #1, and enter body plethysmograph; begin Doppler and EKG monitoring.
1315	Diver #1, lunch.
1430	Diver #1, prepare for #3. Diver #2, out of plethysmograph.
1500	Diver #3, enter inner lock and plethysmograph.
1630	Diver #1, secure plethysmograph. #3 out of plethysmograph.
1700	All divers, parotid study.
1715	All divers, vital signs and 12-lead EKG.
1800	All divers, supper.
1830	Diver #1, prepare plethysmograph for Diver #2.
1900	Diver #2, enter plethysmograph.
1930	Diver #1 and #3, EKG and Doppler.
2000	Diver #1, prepare for #3. Diver #2, out of plethysmograph.
2030	Diver #2, EKG and Doppler. Diver #3, enter plethysmograph.
2130	Diver #1, secure plethysmograph. #3 out of plethysmograph.
2145	All divers, dilate one eye for fundus photography.
2230	All divers, 6-lead EKG.
2300	Taps.

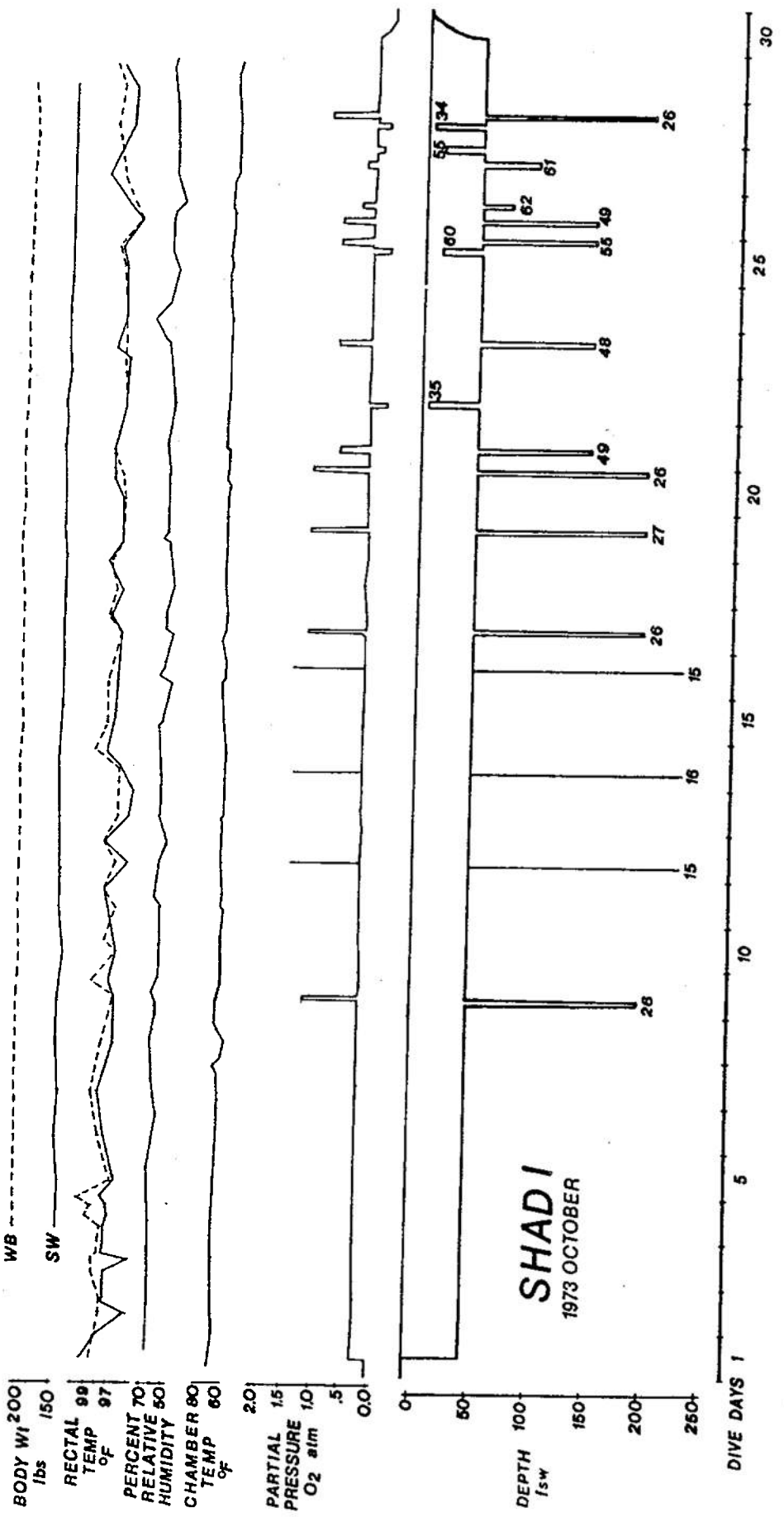


Figure 2-2. Time line for SHAD I. The horizontal axis for all curves is dive days, with Day 1 = 1973 Oct 1. Dive profile reflects simulated depths in dry chamber. Numbers on excursions show duration in minutes of entire excursion from depth until return to habitat storage depth of 50 fsw. Breathing gas was air.

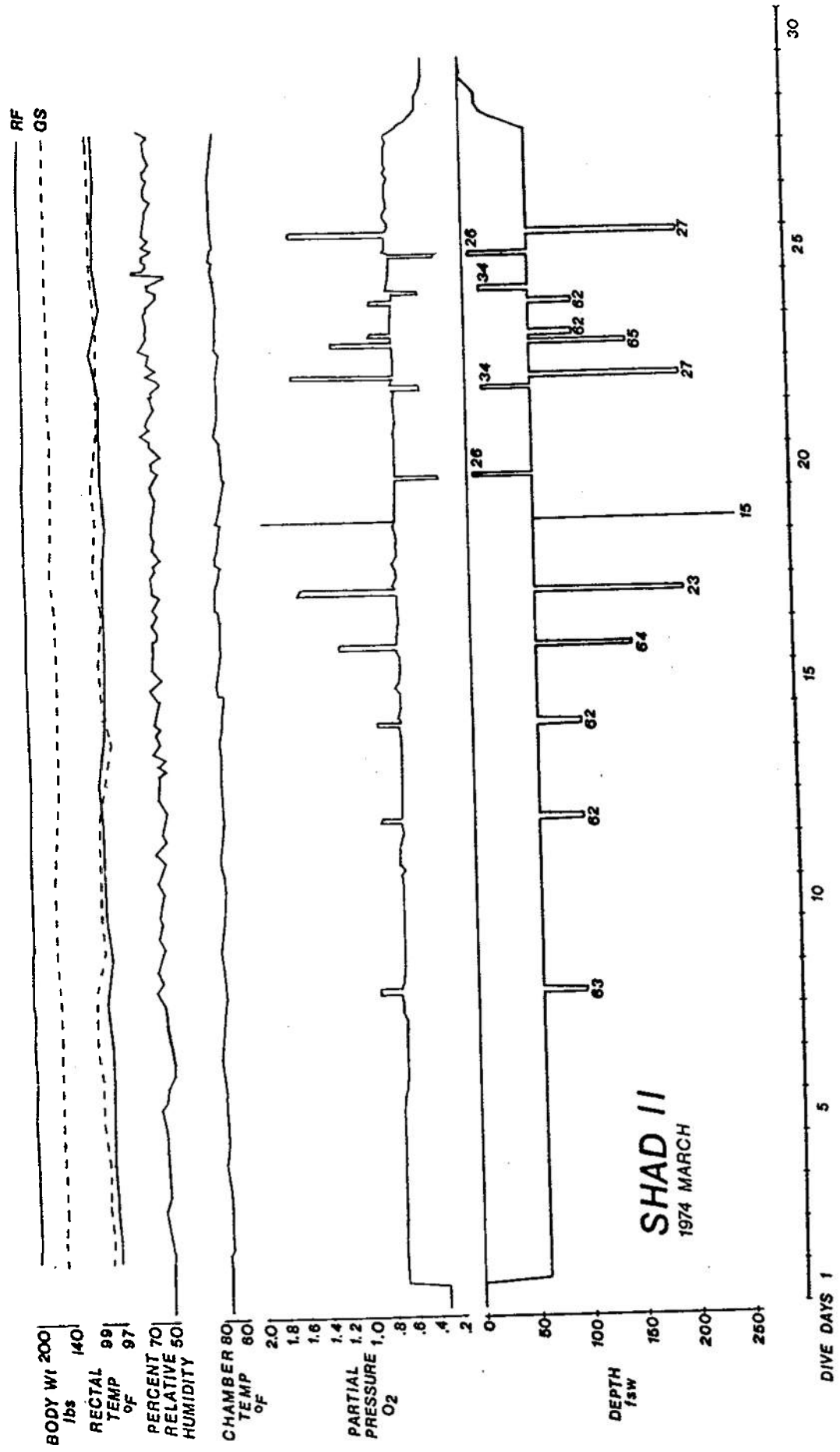


Figure 2-3. Time line for SHAD II. Profile (bottom curve) shows excursions ascending and descending from saturation storage depth of 60 fsw in dry pressure chamber. Numbers show total time of excursion, including travel. Day 1 was 1974 March 15. Breathing gas was air.

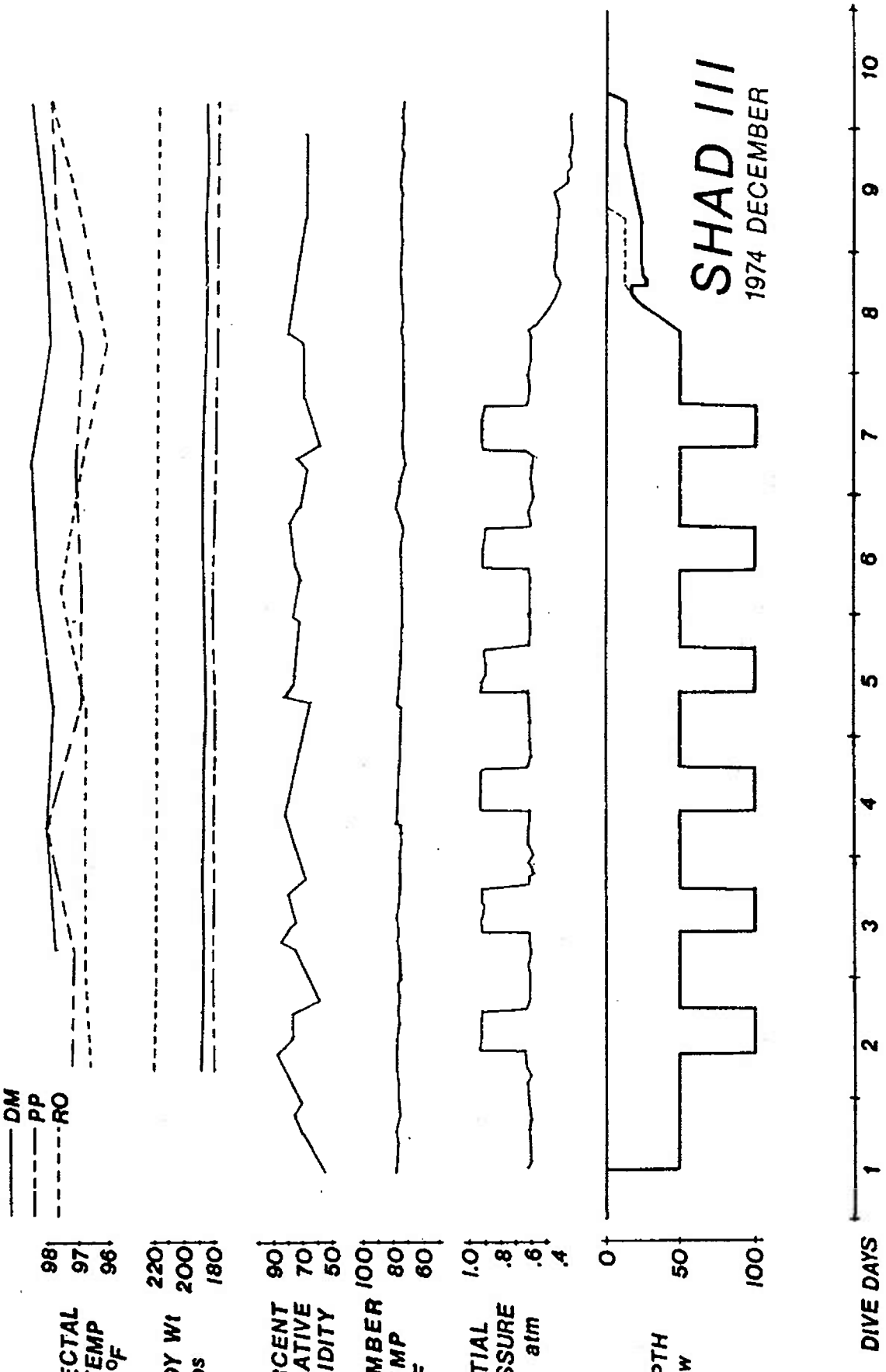


Figure 2-4. Time line for SHAD III. Excursions shown are 8 hr each, from saturation storage at simulated depth of 50 fsw. Day 1 was 1974 Dec 3. Breathing gas was air throughout. The dotted line showed planned ascent, changed because of decompression sickness.

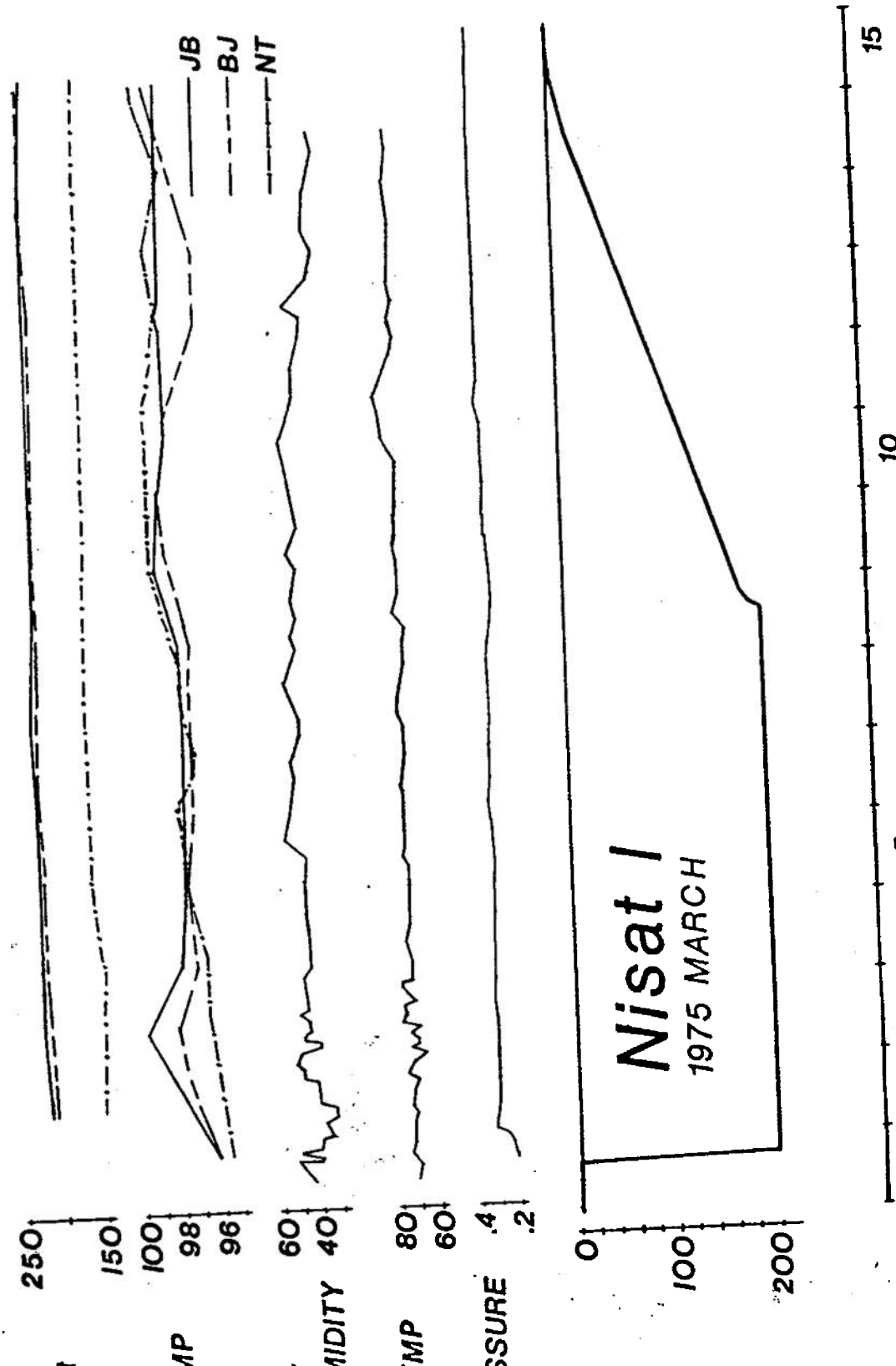


Figure 2-5. Time line for Nisat I. Dive consisted of pressurization to 198 fsw (7 atm abs) with nitrox mixture having PO_2 of 0.22 atm. This was raised after a few hours to 0.30 atm. Day 1 was 1975 March 11. Relative humidity values are questionable.

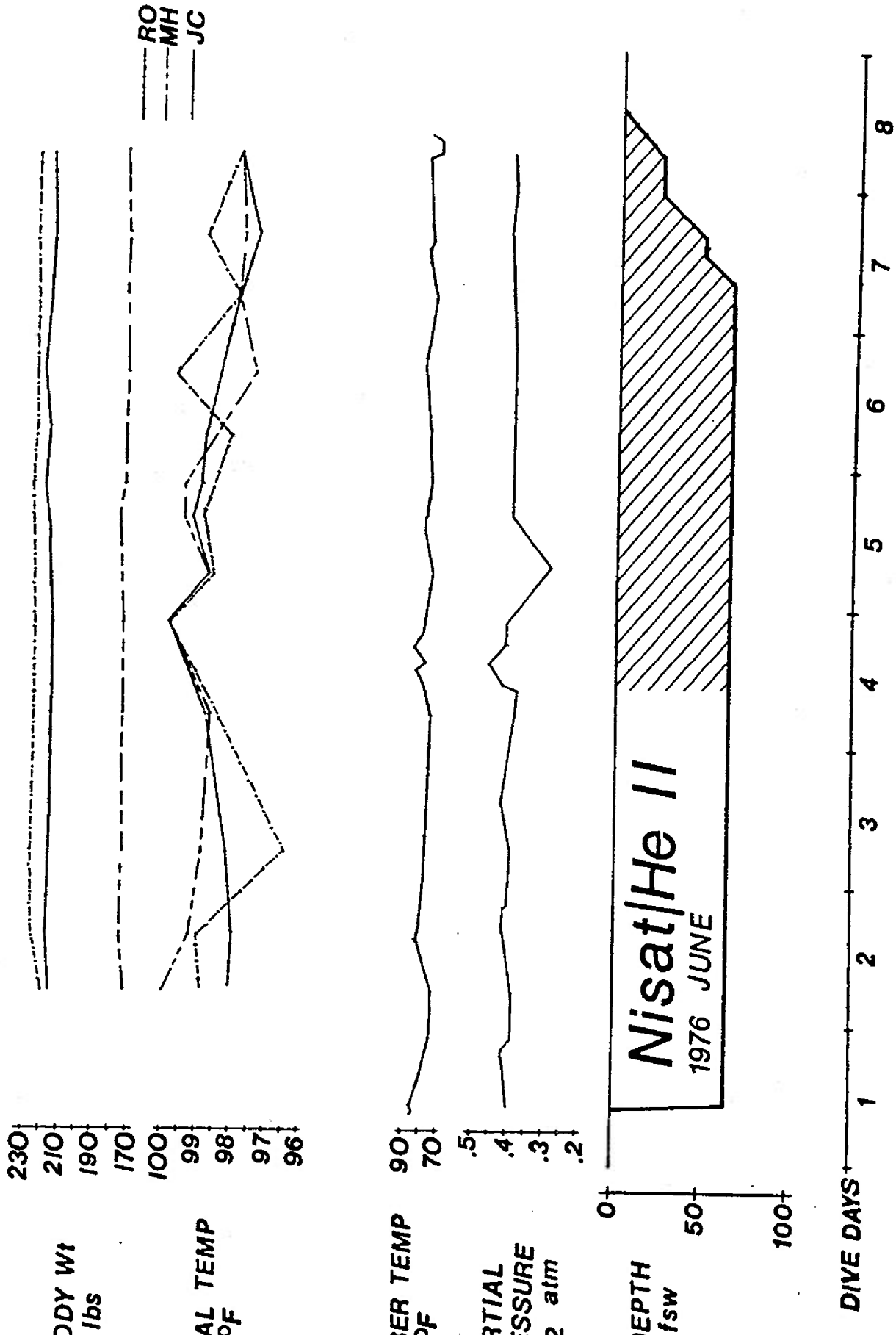


Figure 2-6. Time line for Nisat/He II. Saturation was at the simulated depth of 66 fsw for 3 days with a near-normoxic oxygen level and nitrogen as the inert gas. On day 4 inert gas was changed to helium, shown as hatched area on depth profile. Relative humidity values not available. Day 1 was 1976 June 8.

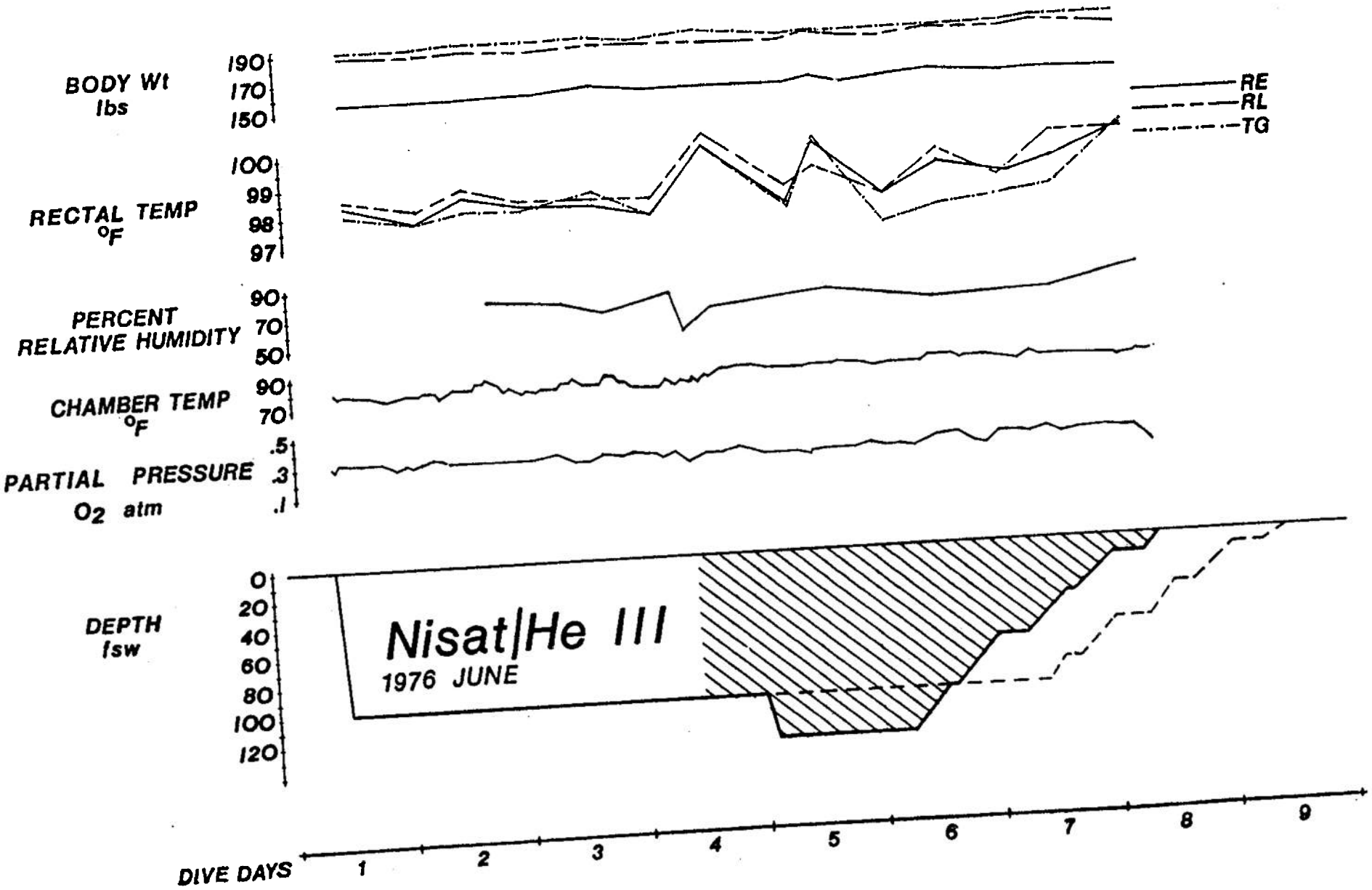


Figure 2-7. Time line for Nisat/He III. Near normoxic nitrogen atmosphere at 99 fsw was switched to helium inert gas at same O_2 level on Day 4. Helium period is shown hatched. The dotted line represents the intended schedule before recompression treatment for counterdiffusion. Day 1 was 1976 June 23.

III.
MEDICAL ASSESSMENT

This chapter reports the overall medical results of the dives. It deals with subjective observations of both subjects and topside crew as well as of the medical officers. Its coverage is broad, overlapping the specific experiments to some extent, and it is spotty, since it relies on available written materials which are not available on all dives in the same degree of detail. While this chapter provides an overview, information fitting the experimental categories such as pulmonary function or decompression is covered, usually in more detail, in those sections as well.

A. Pre-SHAD

The objective of Pre-SHAD was to test the life support and habitability of the chamber complex and to evaluate the 50-fsw saturation decompression schedule. Pre-SHAD consisted of a 2-day exposure to air at 50 fsw. The main finding was decompression sickness in both subjects.

The divers were uncomfortable at first, alternately hot and feeling "clammy." This was eventually resolved by adjustments in the life support system.

During decompression the log reflects that the first vague signs of DCS (decompression sickness) began when the divers were at about 12 fsw, and Doppler bubbles were present in one diver (GA) at that time. He had a dull ache in his right tibia (4" below the knee) and ankle. The pain did not get convincing until he was climbing out of the chamber, at which time he also began to feel ill; he had felt good except for the joint pain before this. The left knee became painful also. After a shower (reported as "hot," considered by commercial divers as not a good idea under these conditions) and oxygen breathing at the surface it was decided that this should be treated. The diagnosis was complicated by the fact that the divers had been exposed to flu before the dive, and GA had flu-like symptoms including a slight fever.

Diver TT had developed left knee discomfort, then pain, at about the same time as GA and with about the same course, and he too had definitive pain by the time treatment was started 3 1/2 hr after surfacing.

These cases resolved on treatment with USN Table 6, leaving slight residual pain which required about two more days to disappear completely. This slow resolution of pain is typical of DCS not treated within a few minutes.

GA came off oxygen (USN Table 6) 9 min before the scheduled departure from 60 fsw because his lips began twitching. He felt some difficulty breathing oxygen by mask during the last ascent from 30 fsw to surface.

After the treatment his chest was tight and deep breathing was painful. This was cleared up by the following morning.

Pre-SHAD clearly demonstrated the need for better decompression procedures, and some improvements were needed in the simulation system, but in the judgement of the medical staff there were no serious medical problems or warnings to inhibit proceeding with SHAD.

B. SHAD I

The SHAD I divers in their 29-day exposure to air at 50 fsw (with excursions) experienced few medical disturbances. The extensive monitoring served to provide a day-by-day confirmation that the divers were doing well. There were no overt symptoms of oxygen toxicity, and no decompression sickness was reported, either from the excursions or the final ascent to surface.

Routine medical and dental problems, predictable for a month-long isolation, arose and were managed. One diver had a boil on his buttock before the start of the dive which recurred during SHAD I. Physiological variations in the biochemical and hematological monitoring were regarded by the Medical Advisory Committee as being not of significance. Psychological adjustments were satisfactory (see IV.D) and considered typical of saturation diving. Hearing threshold downward shifts in the higher frequencies were typical of dense gas environments (Farmer et al, 1971), as were the reduced air movement in pulmonary function tests; these and heart rate changes are discussed in IV.

Exercise capacity was unchanged at depth, but subjectively there was a post dive decrement lasting for about a week. The divers had to stop to catch their breath when walking up a very slight slope. This was interpreted as the result of deconditioning. There were no variations in the electrocardiograms. Rectal temperatures in the 96°F range were recorded but were not considered deleterious.

In general the SHAD I biomedical monitoring did not identify significant deleterious diver variations; the deconditioning was the most prominent effect.

C. SHAD II

SHAD II consisted of a 25-day exposure to air at 60 fsw with 15 excursions. As in SHAD I, a large number of baseline studies were done during the two weeks preceding compression. In addition to those tests which were to be continued routinely during the dive, the subjects underwent ENG testing, vectorcardiography, a long bone x-ray series and participated in NSMRL's Longitudinal Health Study.

Significant medical findings for SHAD II included a drop in red blood cell indexes, fingertip numbness, otitis externa and other skin problems, and evidence of deconditioning during the first few post-dive days.

Diver RF had minor abnormalities in his white cells (a relative lymphocytosis) during the pre-dive period; after a thorough analysis these were attributed to a severe bout with gastroenteritis 2 weeks before.

The daily EKGs gave no evidence of cardiac dysfunction other than rate changes, discussed in IV.C. The daily physical examinations during the dive turned up an expected number of complaints, none of them compromising. These were largely referable to the microbiologic realm.

On the third dive day the left ear canal of both divers was moist, with some redness. By the fifth dive day GS had definite bilateral external otitis infection, and Cortisporin was sent in to be used three times daily. Both divers were cautioned to limit the wearing of the headphones to only those times necessitated by the experimentation since excessive contact with the headphones tended to increase canal moisture, decrease aeration and hence predispose to ear canal infections.

Subject RF did not develop external otitis, but GS persisted in having difficulty. An ear culture on the sixth dive day grew out gram positive cocci. The infection improved some after two days of Cortisporin otic treatment, but recurred bilaterally beginning on the eleventh dive day. Gram negative organisms were cultured from the tap water supply on the eighth dive day and may have contributed to the recurrence which proved stubbornly resistant to Cortisporin otic (which is targeted to the gram negatives). Debris built up in both canals necessitating wire loop removal. Ear powder containing a sulfa drug and chloramphenicol in a steroid base was used rather than the liquid ear drops and dramatic, prompt clearing of the infection was seen. In retrospect, it would seem prudent to have used ear prophylaxis, as has become common practice and was followed in later dives in this series.

RF complained of sore throat beginning on Dive Day 5. There was no associated fever, congestion, headache or adenopathy. On the fourth dive day routine throat culture grew out Candida albicans. The throat was slightly injected but there were no white plaques and no exudate. The sore throat without other symptoms became worse with increased redness, peaking three days after it began with subsidence of symptoms a day later. On the second day of his sore throat another culture again demonstrated yeast organisms. Though there was no clinical evidence for thrush he was treated with Mycostatin. Two days after the sore throat was gone and the Mycostatin stopped, RG again had Candida albicans in throat culture which confirmed suspicions that the yeast probably had little or nothing to do with the etiology of the pharyngitis.

GS also had a mild sore throat on Dive Days 3 through 7 associated with slight congestion. The etiology for the upper respiratory symptoms in both subjects was presumed viral, though it was known it could also represent early O_2 toxic symptoms. In either case it called forth more than the concern usually attending these complaints. If there were early O_2 toxic symptoms they could be a harbinger of worse to come. If of viral etiology, hyperoxia seems to exert no beneficial and possibly some deleterious effects (Gottlieb, 1971).

On the 22nd dive day GS developed gluteal cleft pruritus (itching) and a red rash. Culture of the area grew out Candida albicans and the lesion cleared with use of Mycolog. Both subjects at about mid-dive developed mild angular chilosis (inflammation of the edges of the mouth) which responded to A and D ointment.

An interesting symptom was reported intermittently for 4 to 5 days toward the end of the first third of the dive by GS. He noted a numbness and tingling of his fingers and toes; examinations done during these reporting periods revealed no objective findings and he was not noted during the dive to hyperventilate. These symptoms resembled somewhat a condition noted in divers who have breathed excessive amounts of oxygen during decompression from deep dives. They describe this as if their fingers were stuffed with sawdust. Such symptoms were seen in SCORE (Miller et al, 1976*), and we believe had been observed in the intensive investigations of oxygen toxicity conducted by EDU before WW II, but we were unable to locate a reference.

Rectal temperatures and body weights showed no significant fluctuations or discernible trends from pre-dive through the post-dive period. Food intake was not precisely logged but seemed fairly constant throughout the last three weeks of the dive. During the first week there was relatively greater food consumption.

As the hematology results developed in SHAD II a progressive decline in oxygen carrying capacity of the blood was observed in both divers. This was presumed to be an adaptive response to the hyperoxic exposure, but it was nonetheless incumbent upon the medical team to rule out other potential causes or contributors to the red cell decrease. On the 22nd dive day at a point when subject GS had a hemoglobin of 13.1 and a hematocrit of 38.4 and RF a hemoglobin of 13.3 and a hematocrit of 38.0, the Medical Advisory Committee felt it was time for further tests.

Though there was no evidence of significant bilirubin rise during the dive to suggest the existence of brisk hemolysis, it was felt that chronic low-level hemolysis could be occurring. Larkin et al, (1973) had observed progressive increase in red cell osmotic fragility in 4 healthy volunteers breathing 100% O_2 for only 4 hours. Many other reports exist detailing the relationship between hyperoxia and increased osmotic fragility (see Murray and Jacey, 1977*).

Erythrocyte osmotic fragility, serum iron and total iron binding capacity tests were performed. The % NaCl concentration producing 50% hemolysis was 0.403 for GS, 0.41 for RF and 0.40 and 0.448 for the control subjects, all within normal limits. The serum irons were 117.4 ug/100 ml (ug = micrograms) for GS and 126.0 for RF with the norms for this test being 65-175 ug/100 ml. The controls were somewhat lower, one having 104.34 ug/100 ml and one 52.17 ug/100 ml. The iron binding capacity was 526 ug/100 ml in GS and 578 in RF. The controls had values for this test of 660 ug/100 ml. The higher serum irons and slightly lower TIBC's in chamber vs. control/subjects would fit in with the data of Linmann and Pierre (1968) who showed a progressive decrease in erythrocytic uptake of ^{59}Fe in mice exposed to air at 4 atm for 8 days.

As a further precaution the stools were checked for GI blood loss; in two tests they were guaiac negative. A routine urinalysis done at the same time was without cells in either subject.

Total white blood counts showed a slight decrease during the dive which persisted during the post-dive testing period. This pattern was somewhat in contrast to that seen in SHAD I where there was an in-dive trend toward leukopenia (at the low end of normal) which returned to pre-dive levels by the end of the two weeks of post-dive testing.

There is, from the literature, quite a variable response of white cells to hyperbaric conditions and hyperbaric oxygenation. In Table III-1 are the white cell counts averaged for both subjects and the average lymphocyte and polymorphonuclear lymphocyte (PMN) values for each subject for the particular time period shown. Subject GS, who started the dive with a high lymphocyte count possibly attributable to a preceding bout of gastroenteritis, showed a decrease in lymphocyte count during the dive. Absolute counts were not done. It is noted that during the first week post-dive the average lymphocyte count for both subjects rose. This was associated with a large number of atypical lymphocytes in both subjects. On 4 of 8 blood draws during period E subject GS showed atypicality (9%, 3%, 5%, 5%), and in subject RF two samples showed a large percentage of atypical lymphocytes (9%, 11%). The significance of this is not clear.

A small elevation of the alkaline phosphatase level was seen in both subjects on the morning blood samples prior to compression, and, in general, the in-dive LDH value were slightly lower than the pre-dive values. There was one striking enzyme elevation, this occurring in GS on the 9th dive day. It may be a significant fact that the first excursion, to 100 feet for 60 min, occurred on the 8th dive day. There were no bubbles heard following this excursion and there were no temporally related increases in serum enzyme activity at other times when bubbles were heard. The releasing of tissue enzymes into the plasma in increased concentration is usually a consequence of tissue damage or destruction, but we were unable to establish any consistent relationship of the observed increases (see also section IV.A).

In SHAD II it was noted that the morning glucose samples were slowly but steadily increasing. A predominant effect of hyperbaric oxygen is an inhibition of some of the enzymes involved in carbohydrate metabolism (see review by Hauguard, 1965), but as in the case of RBC loss, other possiblities than merely tissue enzyme inhibition had to be considered. Besides the increase in serum glucose levels there was also a statistically insignificant but discernable downward trend in serum potassium levels. This combination suggested that increased corticosteroid production could be playing a role. The possibility of aldosterone excess was also considered.

The serum glucose increases could have occurred for many reasons other than adrenal activation. We could find no evidence that a glucose rise was typical of a saturation dive exposure. Since O_2 is surely not without effect on every organ system and cell, even the possibility of a direct toxic effect on pancreatic islet cells could not be dismissed. On the 14th dive day GS has a morning "fasting" blood sugar of 155 and RF 149. Consequently, on the following day a 5-hour glucose tolerance test was done, with totally normal results.

Table III-1
White blood cells, SHAD II

Phase	A	B	C	D	E	F
Mean WBC, $10^3/mm^3$	8.9	6.9	7.1	7.8	7.7	7.0
S.E.M.	0.5	0.4	0.3	0.5	0.6	0.5
GS PMN, % total WBC's	45.6	50.0	50.5	50.6	48.9	47.0
Lymph, %	50.9	44.6	42.0	41.1	50.3	46.4
RF PMN, %	62.0	65.6	55.0	57.4	56.1	59.5
Lymph, %	32.4	28.2	38.8	35.4	42.9	31.8

Phases are:
A. Pre-dive
B. Saturation prior to excursion
C. Period of one excursion per day
D. Period of mixed excursions
E. First week post-dive
F. Second week post-dive

The dilemma was resolved with the discovery of candy within the chamber which was apparently being eaten prior to the morning blood draws. While we thought that an understanding of our desire for fasting samples existed, there was a communication breakdown for which the Medical Advisory Committee assumed responsibility. An oft repeated lesson derived from this episode is the dictum to look for the obvious.

Kidney function tests and all urinalyses were within normal limits.

During the SHAD II post-dive period the divers noted a subjective sensation of tiredness and easy fatigability, mostly during the first week following decompression. This was more marked in subject GS. This reaction following SHAD II in both divers was less than that following SHAD I. In SHAD II exercise was a more constant part of the regimen, so perhaps there was less deconditioning. An important correlate of the easy fatigability was the continuing downward trend of the hematocrit and hemoglobin values for first six days following decompression, at which time there was an abrupt, striking reticulocyte increase and upturn in the hematocrit and hemoglobin levels. The post-dive pulse rate increase was mentioned earlier and is discussed in Chapter IV.

By the end of the second week after the dive both subjects reported feeling well. Daily physical examinations and continued blood profiles uncovered no detrimental effects from their 28 day exposure. By the end of the two weeks the hemoglobin and hematocrit were climbing toward normal levels.

One half year following SHAD II subject GS reported no interim physical complaints, however, RF had a left ureteral calculus. It seems unlikely that the dive experience was in any way causative. Serum calcium

and uric acid levels were normal and there was no increase in urine calcium losses. This is in contrast to the substantial bone mineral losses seen during conditions of bedrest and in astronauts incurring periods of weightlessness.

D. SHAD III

SHAD III represented a switch from a diverse excursion pattern to one of a 50 fsw saturation with daily excursions to 100 fsw lasting the duration of a day's work. The exposure was well tolerated.

Distinctive aspects of SHAD III were the development of limitations in pulmonary function in one diver and decompression sickness in another.

A medical report summarizing SHAD III was not prepared, but a "debriefing" by one of the divers (DM) afforded some insight into medical aspects of the dive in addition to the Medical Officer's notes. This subject gave a pint of blood about one month before the dive; another 300 ml were taken during the dive. This is cause to expect some reticulocytosis independent of the exposure itself. DM also mentioned drinking more coffee than was his usual habit, and he admitted eating more, especially during the first few days of the dive (out of boredom). Thus fluid intake was more related to this confinement than to pressure or gas background. His habits returned to normal postdive, so again there could have been an effect not due to the dive per se.

Regarding narcosis, DM feels quite certain that there are narcotic effects at 50 and 100 fsw. He feels there is no decrement in performance, but that there was definitely an effect. He felt further that the tests did not evaluate judgement, and that while performance was not affected, judgement could have been. [It is worth noting that the frivolous behavior observed in SHAD could be as much cause as effect, an effort by the subjects and crew to combat boredom.] DM felt the chamber was surprisingly non-confining, and that the three of them got along unexpectedly well.

Breathing through the pulmonary function apparatus (3 feet of 2" hose) added a substantial resistance, according to DM's subjective feelings, especially at 100 fsw. He felt also that doing the MVV maneuver irritated his throat and tended to reduce performance. DM observed that he could move more air more comfortably by taking deeper breaths and fewer breaths per minute. (Divers find this to be a particularly helpful technique using demand apparatus with dense gas.) He felt his work of breathing at 150 fsw was an appreciable part of the total work load.

DM felt overheated when exercising; the other two subjects considered it less a problem.

Diver DM had a somewhat unusual sequence of chest pains during the six 8-hour excursion days of SHAD III. He acknowledged that he was reporting these symptoms from the viewpoint of a sensitve and perceptive experimental subject, reporting some feelings that might otherwise be ignored. He recovered from a cold before the dive, and did not have one at the time unless it was very unusual.

He felt no pain on compression to 50 fsw or for the first day there. During most of the first 8-hour excursion to 100 fsw he noted "mild deep chest pin and cardiac awareness" that persisted for a few hours after return to 50 fsw. The second excursion day the pain was about the same, perhaps less, but persisted overnight and through the third excursion day. During the fourth excursion the pain increased markedly. It was continuous, but intensified markedly in surges or waves. It felt like trachea and bronchi, and was located beneath the entire length of the sternum and 2 inches to either side. It is described in the log as "softball sized." The pain increased sharply on maximal inspiration. It improved overnight and became appreciably worse the next day; by now it was "fairly painful." The last day it was slightly worse. The pain persisted at this level for about 48 hours, through decompression. It then began to diminish, and by a week was gone most of the time; it was felt during coughing spells for a few more days.

DM had a persistent cough during the same time as the chest pain, and coughing always exacerbated the pain. As mentioned, the coughing was brought on by the MVV maneuver (not an uncommon finding in divers after long oxygen breathing). The chronology of the cough paralleled that of the chest pain, but DM felt some restriction to breathing for a few days after the pains had disappeared. DM suspects but cannot be sure that the heavy breathing during MVV tests may have provoked the chest pain as well as the coughing. It does not do this at the surface to this extent.

DM also developed an inguinal skin rash which cleared after surfacing. He mentioned that he had more mouth sores (several inside each cheek) than usual while in the chamber. These occur more frequently in a "run down" individual. They too cleared after surfacing. DM mentioned the stress of these days, that he was more tired than usual at the end of the day. He noted a metallic taste in his mouth that appeared on Day 6 and faded during decompression. Spontaneous twitching or fasciculation in his thumb was more frequent than usual while he was in the chamber.

The divers' ECG's demonstrated right ventricular conduction delays and diphasic T waves during the first three excursions of SHAD III. Otherwise, the heart rates followed the some pattern of depression during the next three days of the dive and the overshoot during the immediate post dive period as in SHAD I and II (see IV.D).

Diver PP was diagnosed as having pain-only decompression sickness at 18 fsw during decompression. He was treated with hyperbaric oxygen at 28 fsw. There was a transient 24% decrement of his forced vital capacity following the oxygen therapy.

E. Nisat I

Whereas the SHAD dives were unique hyperbaric oxygen exposures, Nisat I was an extreme exposure to hyperbaric nitrogen. The divers were at 198 fsw for 6 days, in relatively normal oxygen.

Two of the three divers were nauseated for the first day, and all three experienced a variety of symptoms throughout the exposure; these include drowsiness, numbness, difficulty with breathing, arthralgia, a "clammy" feeling and otitis externa.

Previous laboratory and open-sea saturation exposures to hyperbaric nitrogen have elicited reports of fatigue, headache, weight-loss, subtle indications of cellular damage, nitrogen narcosis, decrement of task performance, sluggish behavior, electrocardiographic changes, physiological changes, and decompression sickness. Many such problems also occurred during Nisat I. Those which occurred in at least two of the three divers are summarized in Table III-2.

On the last pre-dive day JB had WBC's in his urine but no further evidence of infection. RJ had a high WBC count (15,000) with elevated PMN's and complained of hoarseness two days before, but that had cleared up.

The subjects breathed compressed air by mask during compression to 198 fsw. The 1st diver removed the mask 57 minutes after the start of the 36-min compression; the others removed their masks in turn in the next 12 minutes. Their inspired PO_2 abruptly dropped from 1.47 atm to 0.24 atm upon mask removal. Divers NT and JB experienced the onset of nausea within 2 1/2 hours after leaving the surface. Both subjects noticed the nausea when they were participating in tests of their VER's (visual evoked responses). During repeat tests of their VER's soon after onset of nausea they noted further nausea and the sensations of dry mouth and subjective vertigo. Their appetites were good in between bouts of nausea and vomiting and they ate a full dinner. The nature of their symptoms was likened to the feeling of seasickness, causing them to seek relief by reclining with closed eyes. During this period the divers were purposeful and not particularly giddy. At the same time they were suffering from nausea there were abnormalities in the ECG's of RJ and NT (see IV.C.).

All subjects complained of thermal discomfort fairly early (5 hr) in the dive, describing feeings of "clammy" and being warm or cold. The mean rectal temperature during the bottom phase fell 0.9°F from a baseline value of 98.2°F, supporting the diver's complaints of thermal discomfort at 7 ata in nitrox.

The chamber PO_2 reached its lowest value of 0.22 ata by 6 hours after onset of compression. NT and JB awakened from a half-hour nap 10 hours into the dive feeling less nauseated, but they both vomited again shortly after. The topside personnel intentionally began raising the PO_2 from 0.22 to 0.29 atm at 2355, 11 hours dive time. Diver RJ spontaneously informed topside shortly thereafter that his sense of smell had returned, and it was noted that his speech was quicker and more articulate. After the raising of the PO_2 the ECG aberrancies disappeared and the mean heart rates of all

Table III-2.
Clinical symptoms occurring in at least 2 of the 3 Nisat I divers

Depth fsw	Time of onset*	Time of disappearance*	Problem	Comments
198	4.0		Nausea	Associated symptoms included vomiting, feeling "queezy", dry mouth and subjective vertigo Note: one diver felt nausea two hours after reaching 198 fsw.
198	4.8		Thermal discomfort	Sensation of feeling cold and clammy; "cold sweat"
198	4.7		Somnolence	Strong desire to sleep; fell asleep during human factors testing
198	10.5		Skeletal discomfort	Joint pain and "popping sensation"
198		10.7	Improvement	Temporary relief from nausea, followed by vomiting again shortly thereafter
198	11.0			Began raising PO_2 from 0.22 to 0.29
198	11.5		Abdominal discomfort	Relief by antacids; possibly associated with spicy foods
198		18.5	Improvement	Minimal nausea
198	22.0		Dyspnea	Sensation of heavy breathing
198		40.6	Improvement	No nausea
198	45.0		Headache	
198		69.0	Improvement	No numbness, headache, or nausea
198	72.0		Numbness	Subjects felt "narcosed"; numbness associated with slurred speech and complaint of inability to hold on to objects very well; absence of tremor, weakness and paresthesias.
198		91.0	Improvement	No arthralgia; no numbness.
198	97.4		Exercise tolerance	Felt tired, "low energy level," and exertional dyspnea.
198	136		Otitis externa	Unilateral pain, itching, and swelling of external ear canal.
198		137	Improvement	Last reported episode of numbness
102	238		Skin itch	Transient episode, lasting 10 min
55		280	Improvement	No cold or clammy sensation
50		286	Improvement	No sensation of heavy breathing

* Time is first/last appearance of symptom, in hours after start of compression.

divers fell by 2 bpm. The electrical activity of RJ's heart, who was asymptomatic, did not reveal changes other than of the heart rate. During the time the other two divers were sick RJ, a loquacious individual anyway, was extremely talkative.

Following a second rest period of several hours of sleep (18 hours after compression) the nausea was temporarily unnoticeable. The two divers continued to experience nausea, to a lesser degree, until Dive Day 3.

The consumption of caffeine in coffee was judged to be excessive during the first 24 hours of the dive, necessitating a compromise substitution of decaffeinated coffee, except for the first mug in the morning, after Dive Day 2. Complaints of upset stomach began during the second day. The discomfort was associated with the ingestion of coffee and spicy foods. No occult blood was present in their stools. Symptomatic relief was obtained from antacid tablets. Eventual radiological examinations of the gall bladder and upper gastrointestinal tract failed to reveal any active disease process that might account for the abdominal discomfort experienced during the dive.

All subjects reported the sensation of "heavy breathing" (without orthopnea or ankle edema which might indicate a cardio-pulmonary disturbance) by the morning of Dive Day 2. The complaints of heavy breathing continued throughout the dive until well into the phase of decompression; considering the density this complaint was not entirely unexpected.

Initially, the divers slept as a means of achieving relief from nausea. However, diver JB fell asleep during a human factors test on Day 2. When they slept the divers slept soundly throughout the bottom phase of the dive, as evidenced by difficulty arousing them from naps and nighttime sleep. All subjects felt "slightly drained" on Day 3.

Divers RJ and JB began to complain of headache on Day 3. The headaches were intermittent, not incapacitating, and were accompanied either by dizziness or nausea. Acetaminophen tablets were administered for the headaches. Numbness of the hands and around the mouth was reported by JB on Day 4. NT and JB complained of slurred speech and NT claimed that he could not "hold on to things" too well. These complaints ceased after day 7.

By Dive Day 4, all divers gave the impression that their sense of well being had not deteriorated, and that it had in fact improved compared to their initial problems. At that time they were clearly observed to react slowly to stimuli, and casual investigator interviews revealed that their perception of time was slow. Diver RJ and NT were easily provoked into bursts of laughter and all had the subjective impression that they were "narked." The intensity of the narcosis waxed and waned; the more intense feelings of narcosis were accompanied by slurred speech.

Between Dive Days 2 and 5, divers JB and RJ complained of skeletal problems consisting of shoulder joint arthralgia, audible snapping and pain in the cervical spine, and chest wall pain accentuated by deep breathing and twisting movements. The joint symptoms of arthralgia were suggestive of hyperbaric arthralgia, a condition usually associated with saturation dives to greater depths.

Exercise tolerance tests were begun 97 hours into the dive. The exercise elicited vague symptoms of "low energy level," feeling "tired," and shortness of breath with minor exertion. The divers were able to exercise, but at a relatively low work load. Diver NT aborted the bicycle ergometer test at 125 watts on Day 6 because of undue breathlessness and had to be helped down, and the next day the physician stopped his exercise after a few minutes at 125 watts due to premature ventricular contractions on the ECG monitor. All divers complained of difficulty breathing on the gas exchange apparatus.

Two subjects began local antibiotic treatment for otitis externa during Dive Day 7. Medication consisted of Auralgan and Cortisporin ear drops. Diver NT complained that scratches were slow to heal.

There was a 30 decibel increase of hearing threshold (i.e., a decrease in sensitivity) in the 500 to 3000 CPS frequency range of hearing at 198 fsw. This shift returned to baseline upon return to the surface. Despite this objective finding, JB noted that his hearing perception had changed, and certain sounds such as the compressor pumps sounded louder. This is a common response by divers to acute inert gas narcosis, though objective measurements show a decrement in hearing (see review by Bennett, 1966). Distinct changes were found in the visual evoked responses, visual reaction time, and the electroencephalograms. Although the trends suggested significant alteration of cortical function, a translation of findings into a pathophysiological explanation would be difficult. The clinical hematology examinations did not reveal clinically significant changes of the red blood cell count, hemoglobin concentration, hematocrit, reticulocyte count, platelet count, and white blood cell count as a function of the environment. The clinical chemistry showed elevated levels of serum total calcium, blood urea nitrogen serum total protein, and serum albumin in all men during Dive Day 7. No other trends were noted throughout the experiment. The SGOT and LDH were not significantly elevated in all 3 men simultaneously at any time during the experiment.

During decompression the divers felt less narcotic from 165 to 132 fsw; and the feeling of being "narked" disappeared during the continued transition from 132 fsw to 99 fsw.

The "clammy" sensation disappeared during decompression at around 55 fsw. The symptom of labored breathing was noticably absent at the 50 fsw depth. Diver RJ's ear infection worsened, causing painful throbbing and sleeplessness during the last night of the dive. Transient itching of the trunk and arms was reported by NT and RL at the 102 fsw depth. There were no associated symptoms of pain in their extremities. No signs of decompression sickness were noted at shallower depths or after the dive.

The divers were tired after the dive. Mild exertion brought on shortness of breath. The divers with otitis externa appeared to have mild, partially treated infections. They were able to recall the events of the dive with some difficulty.

Nisat I was a stressful experience, as suggested by the following criteria: (a) the subject's task performances were dramatically reduced at depth; (b) the divers felt distressed in numerous ways; and (c) there were documented medical and physiological changes, such as vomiting, slurred speech, decrement of pulmonary functions, ECG changes, and EEG changes.

The functionally insignificant changes of body weight, respiratory rate, and blood pressure throughout Nisat I suggested that despite the obvious stress, vital body functions were not threatened.

The diver's initial symptoms of nausea, vomiting, desire to rest, queezy sensations, dry mouth, and subjective vertigo were both alarming and fascinating. Quite clearly, this was an unacceptable health status for the predictable performance of work. The clinical psychologists' opinion was that these symptoms were not of psychogenic origin. The environmental conditions may have been causing hypoxia of the central nervous system, or they may have been a response to the combined stressors; the resemblance to HPNS (high pressure nervous syndrome) was sufficient to add support to the gas osmosis explanation of that phenomenon.

Of particular interest were the changes noted in the serial electrocardiograms during the first 8 hours of residence at 7 ata. These may well be explained as vagal activity concomitant with vomiting.

The reduced subjective tolerance for exercise and the PVCs during exercise suggest that working divers may be approaching the limits of exercise tolerance when breathing near-normoxic nitrogen at 7 ata. The diver's complaints of feeling tired and dyspneic with mild exertion immediately post-dive were accompanied by higher than pre-dive values for heart rate. The subjects were apparently experiencing a functional cardiovascular readjustment to sea level conditions, suggesting the advisability for optimal rest periods during the recovery from nitrox saturation dives.

The cluster of symptoms of somnolence, numbness, slurred speech, wide changes in mood, bursts of laughter, distorted concepts of time, and the observed slowing of responses to investigational stimuli were compatible with a state of nitrogen narcosis. The state of narcosis persisted from the initial exposure to 198 fsw through the decompression to 99 fsw. There were no symptoms suggestive of a withdrawal syndrome from chronic exposure to hyperbaric nitrogen, but the withdrawal was quite gradual as it covered several days.

The decompression schedule used for Nisat I was found to be medically safe for the subjects.

The medical problems encountered in Nisat I suggest that under these conditions of human saturation divers were approaching their functional limits with respect to health and the performance of work.

F. Nisat/He II

This dive consisted of a 3-day saturation on normoxic nitrox at 66 fsw followed by an abrupt shift to heliox at the same pressure and oxygen level.

The medical findings were mild somnolence during the nitrogen phase, and mild itching after the switch, without rash.

The divers collectively represented 25 years of diving experience and their age range was from 28 to 35. Diver JC (the oldest and most experienced) was a chronic smoker, as was RO, but MH did not smoke. JC was known to have Type IV chronic hyperlipidemia, and had previously experienced pain-only DCS. MH and RO had resolving upper respiratory infections at the start of the dive.

Mild somnolence occurred in all 3 divers, beginning within 7 hours of reaching bottom. MH and RO complained of "feeling sleepy all of the time." This persisted until after the shift to heliox when they felt more alert and took fewer naps. Duration of sleep was essentially identical in nitrox and heliox, the shortest durations occurring before the isobaric shift and before the onset of decompression (5-6 hr). MH persisted with a non-productive cough throughout the study, and JC developed similar symptoms on Dive Day 5.

The isobaric shift was made by the divers sequentially passing through a zippered plastic curtain separating the inner and outer locks. After the shift from nitrox to heliox, MH and RO complained of generalized itching, but not rash. RO begun itching within 47 minutes of the shift. JC, who had been the first to lock into the helium and had kept busy repairing the curtain and setting up the body box, did not complain of itching until 5 hours after the shift. The itching seemed to predominate in skin areas covered by clothing, and the divers complained of feeling warm. A lukewarm shower seemed to relieve this itching, and all symptoms disappeared by 9 hr after the switch. There were no complaints suggestive of decompression sickness during the ascent to sea level, and Doppler monitoring did not detect any venous gas emboli. The symptoms of itching are typical of "skin bends" and can be considered mild counterdiffusion sickness.

JC received several Rolaids for symptoms of sour belching. He then began ingesting 1 to 4 Tetralac (antacid) tablets a day during Dive Days 2 through 8. JC also began taking Robitussin and Afrin on day 7 for his non-productive cough and runny nose. Diver MH took several Cepacol lozenges on days 1 and 2 for an irritation of the throat. He, too, used Robitussin for a cough from days 2 through 8. All divers used Domeboro ear drops three times a day as prophylactic procedure against the development of otitis externa. In addition, all used 1 to 2 Mydriacil drops in one eye several evenings during the dive as part of the fundus photography protocol.

During Nisat/He II, the pulse underwent statistically significant changes from higher pre-dive values to lower values during the nitrox exposure at depth and from lower to higher values again after the isobaric

switch to heliox. There were no significant changes in blood pressure, respiratory rate, or rectal temperature. Although there was no mean change in body temperature after the switch to helium the daily circadian rhythm became more noticeable (Figure 2-6). Weight showed a rising trend throughout the dive.

Nisat/He II divers manifested clinically abnormal values for the serum triglycerides, cholesterol, SGPT, and calcium. However, the serum calcium, triglycerides, cholesterol, and SGPT were elevated before the divers began their hyperbaric chamber exposure, and the exposure did not intensify the abnormal values. There were single episodes of elevations of the blood hemoglobin, hematocrit, lymphocyte count, reticulocyte count, and blood glucose in JC, the serum phosphorus and total protein in MH, and the hemoglobin and hematocrit in RO. JC's white blood count was slightly elevated on two separate days and his blood urea nitrogen on three separate days. MH and RO had elevated serum albumins twice each, and on different days. MH's alkaline phosphates and SGOT levels and RO's reticulocyte count were slightly lower than normal on several occasions. None of these changes was strong or consistent enough for concern.

MH and RO demonstrated traces of protein in their urine on 3 separate occasions during the dive, but there was no further evidence to explain this as renal dysfunction. MH developed an asymptomatic pyuria; post-dive cultures grew a mixture of gram negative organisms and the clinical impression was an asymptomatic infection of the lower G-U tract.

The Nisat/He II divers complained of mild sleepiness in the nitrox environment at 66 fsw. They responded to the isobaric shift to helium by feeling less sleepy and noticing an improvement in the ease of breathing. The entire exposure appeared to be minimally stressful in view of the stable body weight, the divers' feeling of well being, and the clinically unremarkable changes in their vital signs. The distribution of abnormal laboratory values within the time sequence of events failed to suggest any adverse effects on the body as measured by the blood tests.

G. Nisat/He III

This dive followed the pattern of Nisat/He II except that the saturation depth was 99 fsw instead of 66. The 3 day nitrox saturation phase at 99 feet of sea water in Nisat/He III was completed without incident, but two subjects had occasional joint stiffness and developed headaches the evening of the second day. After the gas shift at 99 fsw the daily body temperature highs were significantly above the earlier values, and the daily circadian pattern became quite prominent (Figure 2-7).

Replacement of the nitrox by heliox in the chamber (day 4) was smoothly accomplished. The subjects began the switch in polyethylene suits that contained nitrox. The chamber gas was then converted to heliox, and the divers made the shift in turn by taking off their suits at 1-hr intervals.

Within the first hour after removing his suit, each subject developed severe skin itching and all 3 developed some form of mild erythematous

rash, and all noted "joint crepitation." No bubbles were detected at any time with the Doppler instruments and the itching experienced by two of the subjects was relieved within 10 minutes when they put the suits back on (no hoods or masks) and flushed them with nitrox. The itching returned within 10 minutes when the suits were again removed. The rashes cleared within the next 2 hours. The itching resolved between 4 and 5.5 hours following the isobaric shift.

Two subjects developed severe headaches the day following the switch; these responded to treatment.

All 3 subjects then experienced some degree of pain and discomfort in the thigh or knee areas beginning between 5 to 7.5 hours after the shift. Two of these had spontaneous resolution of the mild discomfort after 2 to 3 hours. The third subject, however, developed increasing knee pain and active treatment was instituted 12.5 hours after the isobaric shift. Eighty to 90% relief was achieved by a combination of gradual compression to 129 feet (an additional 30 feet) and 6 cycles of breathing a mixture of 50% oxygen and 50% helium for 20 minutes separated by 5 minute intervals of chamber gas (which was 7.5% oxygen and 92.5% helium). All symptoms had resolved by the morning of the 5th day. Final decompression was started from a depth of 129 feet on the 6th day, and there were no medical complications from the decompression.

H. Followup

At the outset of the SHAD program it had been planned to follow the diver subjects for an indefinite period through the Longitudinal Health Study. That program, unfortunately, was discontinued and no further examinations were available through it.

Followup exams of the SHAD I and II subjects several months after the dive showed no abnormalities attributable in any way to the dives.

Two of the subjects have died since their dive, but again there is no known relationship to the dive. SHAD III diver PP died following a fall a few months after the dive. There was some uncertainty as to the cause of the accident, but an autopsy revealed no cause, and no connection with diving was drawn. Nisat I diver NT died of a cardiovascular accident 6 years postdive; he had a family history suggesting a predisposition. An autopsy was not performed on NT.

SHAD I diver JW was rumored to have had a collapsed lung several years after SHAD I, but we were unable to locate him or get or further information as to his condition.

All other divers whose whereabouts are known are thought to be in good health, and no possible long term effects have been noted.

IV.
EFFECTS OF THE EXPOSURES

This chapter pulls together the experimental monitoring carried out on the SHAD-Nisat divers that is not covered in more detail elsewhere. Some topics such as heart function expand on the medical reports of Chapter III. The special "target" topics of narcosis, decompression, and oxygen toxicity are covered later. Much of the material in this chapter has already been published; it is reviewed here with elaboration where warranted.

A. Biochemistry and hematology

The monitoring of the SHAD divers included daily blood samples on which a variety of biochemical and hematological measurements were made. The results were examined and assessed each day by the Medical Advisory Committee. For the most part there were few values outside the normal ranges, and many of these were isolated samples. Some of the outstanding deviations from normal values are pointed out and discussed in the chapter on medical assessment (III).

SHAD I and II blood and urine chemistry showed a number of transient alterations but no consistent trends or differences between the two divers. Total bilirubin was marginally elevated in JW at the start of saturation decompression. LB showed an increase in blood sugar between days 15 and 20, but this we believe was due to ingestion of a small amount of sugar in coffee before the fasting blood draw. Serum total protein, immunoglobulin A and alpha-2-macroglobulin showed no changes throughout the dive (Stewart, 1974*). There was a rise in alpha-1-acid glycoprotein and the 3rd and 4th components of complement at the time the excursion began which stayed up through the postdive period. The significance of these changes is not clear to us.

In a short study of the Nisat I divers' blood, Grober (1975) looked for membrane permeability changes using the red blood cell as a model. Hemolyzed blood from both subjects was found to have the same sodium and potassium concentrations throughout the pre-dive and experimental periods, hence there were no changes detectable by this method.

Hematological indexes during both SHAD I and SHAD II showed an apparent oxygen suppression of erythropoiesis (Murray et al, 1974*; Murray and Jacey, 1977*; Adams et al 1978*). During SHAD I, hemoglobin, red blood cell count (RBC's) and calculated hemoglobin showed a decline during the pressure phase, continuing on down for several days after surfacing. The values went below the normal range at about mid-dive (days 15-17) and became statistically different from pre-dive values during the first post-dive week. During the same period the control subjects showed a slight decline (due presumably to the frequent blood sampling) but did not leave the normal range. Only hemoglobin was statistically different

between the divers and the control subjects. Average decreases were 11.7% for hemoglobin, 12.5% for RBC's and 12.8% for hematocrit.

SHAD I reticulocyte counts remained stable through the 29 days at pressure but rose sharply and became statistically higher than pre-dive during the second to fourth post-dive week; these changes were not matched by the control subject (GS). This is consistent with the concept that the drop in the oxygen-carrying indexes was due to reduction in production of red cells rather than a loss of formed cells, presumably due to a suppression of hematopoiesis by the elevated oxygen levels.

Figure 4-1, shows levels of blood enzymes throughout SHAD I. SGOT (serum glutamic oxalacetic transaminase) is slightly higher than published values throughout in SW, including the predive period. It declines in both toward normal at the end of the compression phase. Without exceeding normal limits, LDH (lactic dehydrogenase) fluctuated throughout the bottom period but not in any obvious pattern related to the excursions. A single increase in SGPT (serum glutamic pyruvic transminase) was seen in SW coincident with the start of decompression. Alkaline phosphatase showed a steady and significant decline throughout the sampling period.

LDH, SGOT and SGPT might be expected to show increases if there were tissue injury, as a result of decompression for example (Powell et al, 1974), or oxygen toxicity. Murray and Jacey (1977*) pointed out that lack of a change in LDH also supports the idea that there was not a significant degree of hemolysis. If there were any effects of decompression in SHAD I they were not reflected in serum enzymes. Alkaline phosphatase excess is an indication of bone metabolic disorders; perhaps the decrease could be expected as a consequence of deconditioning.

The hematological results were even more marked in SHAD II (Murray and Jacey, 1977*) for hemoglobin and RBC's. Diver reticulocytes stayed essentially level during the dive time and first post-dive week, then rose sharply. In the control subjects the increase was more nearly a steady rise throughout the experimental period. Platelets were quite stable through SHAD I's pressure phase, but SW's count rose abruptly at the end of the decompression (from 250,000 to 350,000) and stayed high for at least one month. None of the other subjects showed such a change. Both SHAD II subjects showed high variability in platelet counts.

SHAD III with its daily 8-hours in air at 100 fsw involved a more extreme oxygen exposure than SHAD II, but was for a much shorter period. Even so, red cells and hemoglobin decreased and continued down for several days after the completion of the dive (Adams et al, 1978*). Exsanguination played a smaller role in SHAD III, as samples were taken only every other day and over a shorter period.

Biochemical findings from SHAD III and Nisat I have been reported by Heyder et al (1979*; Tappan et al, 1977*). Perhaps the most prominent finding was a large increase in fluid intake and concomitant excretion. Creatinine clearances were increased in both dives, more so in Nisat I. The increased fluid intake has been seen in other saturation dives and it may well be related to the monotony of the environment. Osmolal clearances were increased during the pressure changes of SHAD III. 17-ketosteroids increased significantly in SHAD III and slightly at the end of Nisat I.

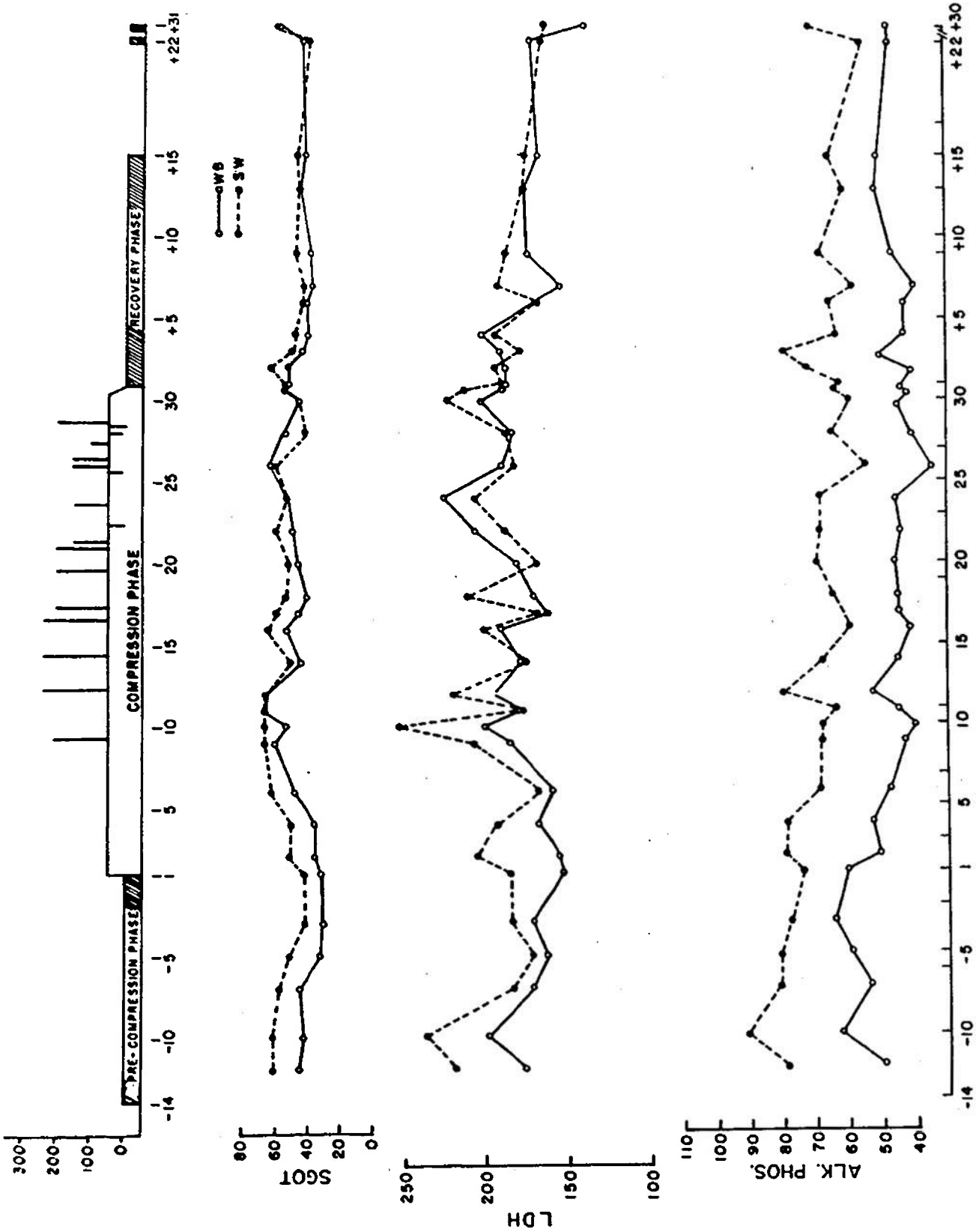


Figure 4-1. Blood enzymes in SHAD I. SGOT was slightly above normal during part of the dive; LDH and alkaline phosphatase showed a slight but consistent decline within the normal range. These values do not indicate tissue injury or hemolysis.

None of the observed changes in serum constituents involved values outside the range of clinical normality, but some of the changes were significant. Serum potassium increased in the latter portion of the dives, suggesting a possible increase in membrane permeability which may be reflected in observed changes in the heart (see section IV.C). Urea nitrogen increased significantly during the hyperbaric exposure and early post-dive period; uric acid was down throughout the study.

During Nisat I the most pronounced serum changes were noted toward the end of the saturation phase and especially during the early post-dive days. These may have been related to the extreme water diuresis seen throughout Nisat I. In contrast to SHAD, the Nisat I divers showed a hemoconcentration after several days at pressure that was relieved on decompression. Cortisol values did not change.

Biochemical changes occurring in Nisat/He II and III are mentioned in the medical section.

B. Respiratory physiology

Of the several physiological parameters monitored in the SHAD-Nisat series, pulmonary function, respiratory gas exchange and exercise are covered in this section, heart function and "vital signs" are in the next, and some aspects of gas physiology are in the chapter on decompression and counterdiffusion (VI).

1. Pulmonary function

Pulmonary function was considered a particularly important parameter to monitor for two reasons, its relation to oxygen toxicity and the effect of gas density. The results of the SHAD series have been reported in detail by Dougherty et al (1978*). Two measurements (FVC and MVV) were made in each test period, and these were conducted 3 or 4 times daily during the dive and at 2-4 day intervals pre and post. Figure 1-8 shows a diver with the wedge spirometer. Six additional determinations were derived from the recordings.

The parameter of interest in monitoring the development of pulmonary oxygen toxicity is vital capacity, in this case forced vital capacity (FVC). No significant FVC changes were seen in pre-SHAD, but measurements were not made at the time the divers were complaining of chest pain. For SHAD I and II there were small changes, scattered throughout the measurement period. Of these a total of 8 samples in SHAD I out of a total of 188 done were more than 2 standard deviations from the control mean; in SHAD II there was 1 out of 170 with this deviation. These changes were scattered, were not all in the same direction, and were within statistical expectations. The conclusion is that FVC did not decrease during SHAD I or II.

There were more definite changes in SHAD III. Diver DM showed a decrease that was cyclical, with the highest daily value in the morning and the lowest after the end of the 8-hour excursion to 100 fsw. The cycles were superimposed on a downward trend that reached a decrement of 7% below control values at the end of the 6th excursion day. Maximum decrement, Dive Day 6, was 9.7%. Diver PP showed an FVC decrease of 23.7% below control values and 27.1% below the late-dive mean value following his relatively light treatment for DCS on Dive Day 9.

Of the 7 SHAD divers (not including pre-SHAD) 6 showed an increase in FVC over the course of the dive; 4 of these were statistically significant.

Maximum voluntary ventilation (MVV) showed no consistent change in pre-post comparisons, varying in both directions by as much as 24%; diver SW showed a statistically significant drop after SHAD I. During the dives the MVV decrements were consistent with the increased density and the data were not subjected to further statistical analysis.

Closing volume determinations were made on the divers in Nisat/He II and III. The lag time to the mass spectrometer introduced variability into the data. The intent was to look for changes in the small lung airways that might result from counterdiffusion bubbles, but the methods did not yield the precision needed for this analysis.

In Nisat I one subject (RJ) showed no change at all in FVC throughout the dive. Divers JB and NT, however, dropped slightly below pre-dive levels during the first 3 days at pressure, then returned to pre-dive levels by the start of decompression. NT showed no further changes but JB showed a consistently higher value during 5 days of post-dive monitoring. It is feasible to explain this temporary drop as being due to gas density, the extra effort of moving the gas. This is reflected also in sharp drops in maximum inspiratory and expiratory flow rates (40-60%) in all three subjects. Maximum voluntary ventilation also decreased (to about 50% of predive) in all three subjects on compression and stayed essentially unchanged throughout bottom time after which all values returned to predive or slightly greater. Another reason why FVC may have been reduced during the first 3 days at pressure was that the divers were ill. FVC requires motivation in order to do properly, and the two subjects with low values may have been lacking in incentive at the time.

2. Exercise and respiratory pattern

The exercise program carried out in SHAD and Nisat I has been reported by Schaefer et al (in press). The exercise regimen used during each run consisted of a 5 minute rest period followed by 10 minutes of exercise at one level and 10 minutes at a slightly higher level and a recovery period. During SHAD the levels were 100 and 150 watts, during Nisat I they were 50 and 125 watts (reduced because of the higher gas density at 7 atm). An open circuit breathing system permitted measurements of volumes by bag collection, and continuous gas analysis was made with a mass spectrometer. Exercise was on a Collins constant work load ergometer, at a pedal speed of 60-70 RPM.

Only 4 exercise runs were performed during the 29 days of SHAD I, and the divers got no other exercise. This resulted in a significant deconditioning, and for this reason more frequent exercise runs were scheduled for the subsequent dives, some in addition to the ones in which measurement were made. Also, the SHAD I data were not included in the analysis because it was felt that the deconditioning would bias the results.

SHAD II did not cause changes in minute volume, respiration rate and tidal volume at rest, but tidal volume increased slightly during the dive. The excursions did not affect these basic respiratory functions.

The effects of exercise in SHAD II were minimal changes consistent with breathing denser gas--slightly larger breaths taken less frequently, with a slight reduction in minute volume and a consequent increase in end-expiratory CO_2. There was deconditioning but it was less than after SHAD I despite the greater loss of red blood cells in SHAD II.

In SHAD III the same respiratory changes were seen, but they were generally more prominent in the measurements made at 100 fsw. There were some slight variations, which are covered by Schaefer et al (in press). These effects are quite consistent with results from other laboratories.

Pulmonary function data from Nisat I show the predicted effects of the increase in density. The was a distinctive drop in MVV, FEV, MEFR and MIFR, of the magnitude expected for this increase in density. There were no noticeable changes during the course of the pressure phase.

Two of the three divers showed no change in FVC, but JB showed a slight but significant ($P<0.001$) drop on going to pressure (mean values 6.2 to 5.8 l) which was stable throughout the bottom time and returned to pre-dive levels after switching. Although his vital capacity was larger than the other two divers he had substantially lower FEV's and his MVV was always slightly lower. Thus the "timed" aspect of the forced vital capacity maneuver in the dense gas may be the explanation; oxygen toxicity is not likely to be involved.

During exposure to the deep nitrox environment of Nisat I there was a marked retention of CO_2, even at rest. Means (rounded) for PCO_2 at rest (sitting) were 32 mmHg pre- and post-dive and 37 mmHg at 7 atm. During 50 watt exercise the sea level rounded mean value was 40 mmHg and the in-dive was 47, while at 125 watts these were 42 and 52, respectively. Whether it was due to CO_2 or not, the stress of exercise was great enough that diver NT was unable to complete two of the runs. The exercise was not started until Dive Day 5, after most of the initial illness had subsided.

Exercise studies were not performed in Nisat/He II and III, and pulmonary function in these dives was as expected. MVV was not done, but inspiratory and expiratory flow rates which were reduced in nitrox returned to normal after the change to helium in both dives. A slight respiratory acidosis was indicated by blood gas measurement performed in the chamber on "arterialized" venous blood, but the results were highly variable (Schaefer et al, 1977*).

C. Cardiac function

1. SHAD I, II, III, and Nisat I

Observations on heart function of the SHAD and Nisat I dives have been reported by Wilson et al (1977*). A discussion of the methods is included there. Essentially this involved daily 12-lead scalar electrocardiograms, using ink markings on the diver's chests to ensure correct and comparable recordings from day to day. Divers were supine.

The most obvious and prominent effect seen was a reduction in heart rate during the pressure phases of all four dives. In SHAD I and II it began immediately on compression with a drop of 15-20% (70 to 56 beats/min), followed by a gradual recovery to pre-dive values after one week of stable saturation. During the excursion phase the rates were reduced following each descending excursion, and increased slightly or remained about the same during the ascending ones. Most of these changes were statistically significant. The rates were taken from the ECG recordings.

At the end of both SHAD I and II the heart rate went up for a few days to levels significantly higher than pre-dive, coincident with the observed period when the deconditioning was most obvious.

The corrected Q-T interval (Q-Tc; the "correction" is to adjust this interval to be independent of heart rate. Bazett, 1920) taken from the ECG tracings showed a pattern of reduction similar to that of heart rate for these two dives. No significant changes were seen in P waves, P-R interval, rhythm, QRS or .T vector, QRS duration, ST segment, T waves or voltage. All tracings showed normal sinus conduction.

Vectorcardiograms were done in SHAD II to serve as baseline; they could have been performed in the chamber if warranted. It was planned to do them in-dive to give a possibly more complete cardiac evaluation if any deviations were seen in axis, intervals, rhythym or voltage of the daily scalar EKGs. Acute exposures to hyperoxia had been shown to cause a negative inotropic effect or decreased myocardial contractility in experimental animals, so it seemed prudent to follow cardiac status carefully. Chronic hyperbaric O_2 at SHAD III levels could conceivably cause EKG changes reflecting myocardial toxicity. This could be monitored using vectorcardiograms, so these were done pre-dive to serve as a baseline.

SHAD II vectorcardiographs were performed with a Hewlett-Packard 1507A vector programmer, 1508-11A lead networks, and a 780-6A Viso-scope; they were analyzed using the trigometric method (Beckwith, 1970). By comparison with standard reference texts the calculations were all within normal limits. These included the direction of vector rotation, the millivolt, and angle of the P, R and and T waves, and the 0.02, 0.04, 0.06, and 0.08 second mv and angle of the QRS loop.

On the second day of SHAD III during the first excursion to 100 fsw all three divers demonstrated a slight right ventricular conduction delay characterized by rSR' complexes in the right precordial leads and diphasic T waves. Rates fell on reaching saturation from 73 to 58 bpm, then to 53 on the first excursion. After the first excursion the rate stayed about the same level, but on subsequent days the excursions caused significant depressions from a gradually rising baseline at 50 fsw. By the end of the week-long experiment the mean rate was no longer reduced below pre-dive values.

During the first 8 hours of Nisat I diver JB (one of the two who got sick) showed a lengthening of the P-R interval and a progressive flattening of the P wave until it was no longer visible. He also showed a minor ST elevation that remained within normal limits, rSR' complexes in the precordial leads, and a diphasic T wave. After the oxygen level was raised the rSR' and T wave changes disappeared and the P wave reappeared but in a flattened form. At no time was the flat or square wave S-T depression noted that would be indicative of myocardial ischemia. Diver NT showed occasional premature ventricular contraction and peaking of precordial T waves; the PVC's disappeared after oxygen was raised and the P wave became more demonstrable, but the T-wave changes persisted. The asymptomatic diver (RJ) showed no ECG changes through this period (other than rate).

Blood pressure, both systolic and diastolic, dropped steadily throughout Nisat I; for example, the mean systolic pressure was 136 predive, 131 at 198 fsw, 129 during decompression and 125 during the first few post-dive days.

The Nisat I mean rate changes were similar to the SHAD dives, with an initial drop from 72 to 58, a return to 69 by the beginning of decompression (but still significantly below pre-dive) and an overshoot to well above pre-dive after surfacing.

In exercise 2 of the 3 SHAD III divers showed occasional premature atrial contractions at the 150 watt work level (Schaefer et al, in press*). During exercise in Nisat I divers NT and JB both had PAC's at the 125 watt load, and NT had episodes of PVC's which caused the exercise to be terminated by the physician on Day 7. NT was the smallest of the 3 at 161 pounds.

Most of the effects seen in these dives have been observed in other somewhat similar circumstances. Flattening of the P wave, nodal rhythms, idioventricular rhythms and PVC's have been noted in apneic immersion studies which elicit the diving reflex. The repolarization changes and the right ventricular conduction delay are suggestive of electrolyte disturbances in the myocardium, but there were no clinically significant shifts in blood electrolyte levels.

The bradycardia, nodal rhythms, peaked T waves, and premature ventricular contractions may be explicable by reflex, vagal nerve activity. This is especially likely in the divers who were nauseated; during periods of vomiting vagal responses can completely obscure the normal ECG. The right ventricular conduction delay can apparently occur in either hypoxia or hyperoxic, hyperbaric environments. The shortened Q-Tc intervals may be dependent on hyperbaric air.

These experiments demonstrated a strikingly consistent example of hyperbaric bradycardia, reported first by Shilling et al (1936), and as yet not fully explained. Some of the response as seen here has been partitioned (Hesser et al, 1978) into effects of oxygen (as an increase in vagal tone and as a direct myocardial action) and nitrogen (as a beta blockade), but there are also direct pressure effects (Ornhagen, 1977). Other possibilites include thoracic receptor effects due to breathing dense gases, temperature, and electrolyte shifts.

2. Nisat/He II and III

Standard 12-lead EKG's were made at frequent intervals during the Nisat/He dives. These were done while divers were at rest, supine, in a near basal condition before getting up in the morning, and again just before the evening meal.

The results of an analysis following standard methods (described by Kossmann, 1953) are given in Table IV-1. The values presented are mean and standard deviation for each of the parameters P, QRS, SV_1, RV_5V_6, axis, QTc, PR and heart rate. The phases of the dive are I Pre-dive, II Nitrox exposure, III Heliox exposure, IV Decompression, and V Post-dive. Wave amplitudes are given in millimeters, on a scale of 10 millivolts/millimeter. An increased T-wave would be an indicator of atrial hypertrophy which might result from restriction in the A-V valve. The PR and QRS intervals are indicators of changes in ionic balance or drugs.

A look at Table IV-1 shows no remarkable or consistent changes. The classical bradycardia (Wilson et al, 1977*) is seen in Nisat II but does not show in Nisat III under quite similar environmental conditions. This is typical of this phenomenon which as mentioned above -as yet to be satisfactorily explained. The method of lumping the data has perhaps obscured some of the information. For example, in Nisat/He II a reduction in heart rate during the nitrox phase is seen, but 2 of the 3 subjects had values on the second dive day several beats lower than at any other time in the dive. The same thing is true for 2 of 3 Nisat/He III subjects, but the differences were smaller. Thus 4 of 6 subjects showed bradycardia in hyperbaric, near normoxic nitrogen during the first 2 days, and the same 4 showed a trend toward an increase after the switch to helium.

Thus it appears that except for the bradycardia, the nitrox exposures and shift to helium did not have significant effects on cardiac parameters as measured by the EKG. There were no signs of deleterious changes.

Table IV-1. Analysis of ECG for Nisat/He II and III

Nisat/He II

PHASE	P	QRS	SV_1	Rv_5v_6	Axis	Q-Tc	PR	HR	
I	1.1	0.09	9.1	14	22	0.40	.13	67	(mean)
(n=18)	0.3	.01	2.3	2.1	21	.02	.01	8.4	(sdev)
II	1.1	0.09	9.1	15	25	0.40	.14	62	
(n=15)	0.5	.01	1.5	2.1	18	.01	.01	6.5	
III	1.0	0.09	9.1	15	21	0.41	.13	63	
(n=15)	0.3	.01	1.8	2.0	18	.02	.01	8.8	
IV	1.0	0.09	8.7	15	20	0.40	.14	60	
(n=6)	0.3	.01	2.0	2.1	20	.01	.01	7.2	
V	1.2	0.09	9.0	14	22	0.41	.14	69	
(n=18)	0.4	.01	1.8	2.2	19	.02	.01	6.4	

Nisat/He III

PHASE	P	QRS	SV_1	Rv_5v_6	Axis	Q-Tc	PR	HR	
I	0.7	0.08	8.0	22	56	.39	.15	51	(mean)
(n=27)	0.4	.01	1.7	8.0	22	.01	.02	4.6	(sdev)
II	0.7	0.08	8.2	24	58	.39	.14	51	
(n=15)	0.5	.02	1.8	7.6	19	.01	.01	4.7	
III	0.8	0.08	8.4	23	54	.41	.14	57	
(n=15)	0.5	.02	1.7	8.0	21	.02	.01	7.4	
IV	0.7	0.07	8.4	23	54	.39	.14	56	
(n=12)	0.4	.01	1.7	9.5	20	.02	.01	7.1	
V	0.9	0.09	8.0	21	47	.42	.16	58	
(n=12)	0.5	.01	1.7	6.0	30	.01	.01	8.4	
Normal range	0 3	.03- .11	5.0- 25.0	5.0- 28.0	-30°- +120°	0.3- 0.43	0.11- 0.21	40- 100	

Phase:
I = 6 predive days
II = Compression and 3 days nitrox saturation
III = Heliox saturation, 3 days
IV = Decompression, 2-3 days
V = 2-3 postdive days

Amplitude = 10 mv/mm
Paper speed = 25 mm/sec
n = number of measurements included in analysis

Parameter:
P = P wave amplitude, max of leads I-III, mm
QRS = QRS interval, max of leads I-II, seconds
SV_1 = S wave, max in lead V_1, mm
RV_5V_6 = R wave, max in V_5 or V_6, mm
Axis = Mean QRS vector, degrees.
Q-Tc = Corrected Q-T interval (Bazett, 1920)
PR = PR interval, difference between max P-S interval and max QRS width, leads I, II, III, seconds.
HR = heart rate, beats/min

D. Psychology and behavior

R.W. Hamilton, B.B. Weybrew and E.M. Noddin

Behavioral studies are found here and in the chapter on narcosis, V. This section deals with the ability of the subjects to cope with the experimental exposures and Chapter V deals with the effects of the environment on their performance.

1. SHAD I

A comprehensive program was set up for screening and selection of the subjects in SHAD I. This is discussed in II.C. and covered in more detail in a report by Weybrew (1974*). Psychological assessment in SHAD I and II was "non-intrusive," in that there were no daily psychometric ratings as was traditional for this laboratory (see, for example, Weybrew and Noddin, 1971). Instead, the method used was direct observation of the subjects, periodic examination of the diving log, and pre- and post-dive interviews. These observations were interpreted in the context of the personality profiles constructed from the diagnostic test data. The pre-dive examinations are discussed in II.C.

The post-experimental diver interview schedule was designed to provide very general probes so as to obtain approximately the same interview responses from all diver-subjects. It was divided into the following five parts: (1) general interview probes, (2) symptoms peculiar to the chamber situation, (3) symptomatology related to gaseous and/or pressure conditions, (4) immediate psychological effects of "surfacing," (5) abbreviated guidelines for a brief mental status examination.

At the end of SHAD I it was felt that the basis for recommending the two experimental subjects (out of the 4 diver volunteers) was essentially correct. These selection criteria were: (1) personality profiles on the MMPI (Minnesota Multiphasic Personality Inventory) most congruent with MMPI normative data obtained from samples of Navy divers; (2) approximately equivalent in verbal aptitude, similar interests, similar motivation for volunteering, and a friendship history; (3) no evidence of serious neurotic or depressive trends; (4) no history of emotional or adjustment problems; and (5) relatively more deep diving experience.

There were no perceptual changes reported or observed other than one diver reported voice communication seemed attenuated; visually, the "closeness" of the chamber remained unchanged.

Their self-observations regarding their own and the other diver's performance on the various tests and procedures suggested that performance remained the same or improved with practice, except when their motivation to carry out certain tasks declined remarkably and/or attitudes toward certain of the experimenters deteriorated.

Both men's attitudes regarding the significance of certain tests and procedures deteriorated considerably about midway the experimental period. Further, any modification in the scheduling of tests tended to exacerbate the negative reactions. The two divers' demand for "structure of events" as time went on increased greatly. The imposition of two unscheduled data collection routines on the day before surfacing led to rather intense emotional outbursts and negative attitudes toward the experimenters.

Finally, both men were disturbed by the lack of structure of events after surfacing. In sum, stress exposure seems to enhance the need for structuring environmental events.

Somatic symptoms which might have been correlated with changes in emotion were infrequently mentioned in the interview. While neither man complained of reduced quality of sleep, both emphasized insufficient quantity of uninterrupted sleep. Possibly the most consistently mentioned symptom was fatigue. Boredom, sleep deficiencies, confinement, deprivation of various kinds, and lack of exercise are all fatigue-inducing factors, but whether there are any atmospheric-related factors contributory to fatigue was not determined.

Excursion dives were anticipated as a means of speeding up passage of time. Both became really "narked" on one dive only, namely the 200 fsw dive on 9 Oct 1973, while the other deeper excursions (they said) did not produce these effects. They were unusually tired prior to that particular 200 fsw excursion. Regarding work on excursions, the divers said if the tasks were meaningful diver activity (and not experimental tests), they felt they could and would perform them effectively.

On the basis of a brief Mental Status Examination given at the end, there was no evidence of thought disturbance of any kind in either man. The affective signs usually associated with refief, or what might be called the "end effect" (elation, verbosity, hyperexcitability), were not observed in either diver during the post-experimental interview 20 hours after surfacing, though they were observable earlier. Instead, a moderately depressed mood state, somewhat typical of anticlimatic situations was obvious in both men. Sensorium was clear with no evidence of disorientation (time, place or person). Tests of both remote and recent memory were unchanged from pre-experimental data. No motor abnormalities were observed. In short, there were no differences of any significant magnitude noted in comparing the pre- and post-experimental MSE observations.

2. SHAD II and III

Details regarding pre-dive psychological assessment of SHAD II divers are not available; however, one subject (GS) was one of those not chosen for SHAD I. There was an altercation between divers GS and RF during SHAD II that is not reflected in the logs. The basis of it was continuous ribbing regarding diving experience, but no details are available. A pre-end-effect moodiness (depression-like but not true clinical depression) was noted in SHAD II and III, about halfway through decompression.

Impressions from Diver DM during SHAD III are given in the medical report. This and subsequent dives were much shorter than SHAD I and II and therefore led to less long-term stress.

3. Nisat I

Nisat I may have been the most interesting environmental exposure of the session; it was the subject of psychological studies leading to a published report (Weybrew, 1978*; in that report Diver A is RJ, B is NT and C is JB).

Using the same normative data from a group of typical Navy divers the MMPI pre-dive scores showed this group of 3 divers to be remarkably similar to each other and not statistically different from the normal group. There were individual differences in specific areas of the MMPI which have been correlated by Weybrew (1978*) with some of the observed responses. All three of the Nisat I divers were judged to be slightly extroverted, to handle emotions outwardly, and to have a low probability of depressive or anxiety reactions of any sort. These pre-dive assessments were generally borne out by the divers' response to the experiment.

One test of a "performance" nature was given twice a day (at reveille and taps) to the divers along with the Multiple Affect Adjective Checklist (MAACL). The performance test measured sequential reaction time by having the divers cross out upper case C's mixed with O's, and the adjective checklist assessed mood. No structured debriefing was conducted after the dive. One diver commented post-dive that he felt he had done well on the early tests, but results showed otherwise.

Results showed in general that reaction time increased substantially (indicating a performance decrement) just after compression, then showed a recovery to predive levels over the next two days. There seemed to be another reaction time increase after that, and again a decline back to below (i.e., better than) pre-dive scores by the end of the pressure phase. The mood indicators changed in a somewhat parallel way, with hostility, anxiety and depression showing an increase after going to pressure. Scores continued to improve during decompression. The second phase of reduced reaction time, about 48 hours into the dive, was associated with a period of general fatigue. This may have been a sequela of the 2 days of illness. Diver JB was particularly affected by the fatigue and drowsiness. It took about 4-5 days for the divers to completely adjust to the 7 atm nitrox environment and be capable of considerable useful work.

An attempt was made to explain the nausea experienced by the divers on the basis of the personality and mood data. Divers NT and JB began to vomit about 3 hours into the dive and remained in and out of a nauseated state for the next 44 or so hours. Diver RJ did not vomit but did feel some abdominal distress. NT and JB were sleeping during the time (about 10 hours into the dive) when oxygen was added. They felt better on awakening, RJ felt fatigued, and the reaction time scores reflected this, slightly.

One possible reason for JB's fatigue is that he was a coffee drinker, and the divers had been switched to a decaffeinated coffee on Dive Day 3; however Diver RJ drank much more coffee than JB and he was apparently not affected by the switch.

There were slight changes in the mood index (MAACL) during the nausea phase, but it appears more likely that these were as a result of the nausea than a cause of it. Also, Diver RJ showed less decrement on reaction time scores than the others during this period.

Review of a list of causes of nausea (Resnick, 1966) reveals nothing that can explains the nausea seen. It is known to result from psychogenic causes (Grinker and Spiegel, 1945), but the mood scores reveal no deviations (e.g., high anxiety) of sufficient magnitude to explain the results by that (see V.B.).

E. Sensory and brain function

1. Visual function and EEG

The visual functions studies covered included Ortho-rater acuity testing, field of view, night vision, color vision, fundus photography, visual EBR's and EEG's (Kinney et al, 1974*).

The Ortho-rater is a device for mass screening, measuring monocular and binocular acuity under controlled lighting. It also test phorias and depth perception. Visual fields were tested using two spherical targets, one fixed and one movable. Night vision uses the NSMRL test (Kinney et al, 1960) which uses 120 small lights in the visual field of a dark-adapted subject. Fundus photography was used to determine the degree of dilation of the retinal arteries and veins in a response to the hyperbaric oxygen. Visual EBR's (VER's) were taken from an electrode located over the visual cortex, recorded on tape, and processed later through a signal averager. Two stimuli were used, a pattern of vertical stripes, or a plain white light covering a porthole, flashed at one or 16 times per second. One hundred recording intervals of one second each were used for each measurement. Color vision was assessed with the Farnsworth-Munsell 100-hue test. The same electrodes were used for recording EEG after the VER run was completed.

Vision tests occurred every 3 or 4 days during SHAD I, less often during SHAD II. Control studies were run several times before and after each dive.

There was very little change in the visual acuity of the divers throughout SHAD I and II. If anything, acuity was slightly better during the dive. Night vision and depth perception also showed no changes. Fundus photography showed a constriction of both arteries and veins with compression which did not change throughout the dive and which recovered promptly after the dives. VER's showed changes, some of them typical of normal variations. At 50 fsw the main effect was greater variability. At

60 fsw, the effect was likewise more an increase in variability than a decrement in performance. On the excursions to 200 fsw, however, several subjects showed consistent VER changes typical of those seen on deep air dives. Changes in the EEG, mostly increases in alpha waves, were variable during the dive and were not well correlated with the ongoing event; they were not typical of pre-convulsive activity. Errors in color vision during the dive were higher than controls, but this appears to be satisfactorily explained as being due to lack of motivation.

In summary, in SHAD there was no evidence of deterioration in visual performance.

During Nisat I these same measures were made, plus reaction time. They showed changes not easily attributed to the known stress but that are typical of general CNS dysfunction. It is reasonable to attribute this to narcosis. Interestingly, with the exception of reaction time the greatest detriments were on the 3rd or 5th day rather than the first when the divers were most seriously affected (McKay et al, 1977*).

2. Electronystagmogram, SHAD II (R. Williamson)

The electronystagmogram, (ENG) performed as baseline, could also be done in the chamber at depth if needed for a thorough evaluation of any complaints referable to the vestibular system. Because O_2 toxicity shares symptoms with those produced by vestibular derrangements it was felt important to have ENG capability to help differentiate the two during the dive. Decompression sickness can also be manifested solely as inner ear disease without other symptoms. Because DCS can affect hearing as well as the vestibular apparatus, audiograms were also part of the medical routine.

The ENG's were limited to ice water caloric stimulation of both ears with the patient lying flat and his head tilted 30° to the horizontal. A fine flexible plastic catheter was inserted into the canal and 25cc of ice water was injected over a one-minute period. The subject kept eyes closed during the entire test and was instructed to count backwards from 100 by 3's to prevent central inhibition of nystagmus. Five minutes after one side was tested the other canal was irrigated.

Conventional EEG electrodes and conductive gel were used to record the corneo-retinal potential with the anode at the right outer canthus, the cathode at the left outer canthus and the indifferent electrode placed in the midline of the forehead between the eyes. Calibration was carried out by having the subject switch gaze between two points which are so placed that they subtend an angle of 10 degrees to the glabella of the subject. The recordings were made on a Gould Brush 8 channel strip chart recorder and were continued until the nystagmus dissipated.

Post-dive ENG results showed a slight bilateral canal hypofunction in RF; this diver had been hospitalized for a vertiginous condition that was not diving related for two weeks 3 years previous to SHAD II. There were no vestibular problems or distinctive findings after SHAD II in other subjects.

F. Oral biology

Salivary samples were taken on all the dives. Sampling was done with a disk custom made to fit each diver over the parotid duct and was held in place by a ring of gentle suction around the duct.

Biochemical findings on saliva were generally unremarkable and have not been written up for publication. One experiment which has been reported considers the effect of the high oxygen environment of SHAD III on parotid secretions.

Acute hyperoxia has been reported to inhibit transepithelial transport in frog skin and toad bladder, and to produce histologic changes in rat parotid gland (Houlihan and Down, 1972). These findings raise the possibility that transport inhibition may play a significant role in the development of O_2 toxicity in man. Miller and Esquire (1978*) studied transepithelial (parotid) transport in 3 subjects exposed to chronic hyperoxia (mean PO_2 0.61 ata) for 7 days during SHAD III.

In these studies, marked inhibition (up to 60%) of stimulated secretion developed progressively during hyperoxia, with recovery thereafter. From the relation between flow rate and secretion rate, it was possible to estimate the contributing factor of acini and ducts to the final secretions. By this approach, it was calculated that the primary secretion had a constant composition, with Na=60 and K=18 mEq/L. Ductal reabsorption of Na had a transport maximum (Tm) of 15 mEq/min, and the primary secretion rate varied wih the externally measured secretion rate.

These data support in general the 2-stage secretion hypothesis, but are not consistent with the view that the primary secretion is plasma-like. By this analytical technique, the site of the observed transport inhibition is concluded to be an effect on the primary (acinar) secretion rate.

O_2 is directly implicated as the causative agent by the finding of further parotid inhibition in one subject receiving 100% O_2 for bends treatment during decompression, and by concurrent findings of pulmonary oxygen toxicity. Hence, parotid transport inhibition is considered to be a new early indication of O_2 toxicity in man, and a component of that toxicity as well, and to raise the possibility of toxic effects in the other, less accessible transporting epithelia.

It can therefore be postulated that inhibition of parotid secretion is a manifestation of oxygen toxicity. Recovery times were not determined, but this technique might be used to monitor recovery of the lungs under varying conditions. It also raises the possibility of toxic effects in other transporting epithelia. These experiments as they were performed here do not rule out the possibility that the effects seen were due to a transient and reversible vasoconstriction similar to that seen in the fundus of the eye (IV.E).

V.
NARCOSIS AND EFFECTS OF HYPERBARIC NITROGEN

The narcosis and breathing resistance aspects of the nitrogen in air are well known to the diving community. The SHAD-Nisat series gave an opportunity to look at the effects of longer exposures than those normally experienced in air diving. Of greatest interest here are the effects of living in a hyperbaric, nitrogen-based atmosphere, and whether any "adaptation" results from this residence.

Two aspects of the nitrogen-based atmosphere are considered here, its narcotic properties as they affect performance, and that combination of properties which resulted in the illness of the Nisat I divers.

A. Human factors/Performance

Human factors performance tests were given on all dives in the series. These are in general derived from established tests, but one of them forces the subjects to work near the limit of their ability in the control condition as well as during the exposure. These have been described by Moeller (1975*) and in other reports from NSMRL (see Moeller et al, 1981). The tracking task requires the subject to follow a moving dot on oscilliscope screen by means of a "joystick," and gets more difficult as the subject's skill improves.

Other tests were arithmetic, 5 choice reaction time, short-term memory, cognitive tests, sequential reaction time, time estimation, and a mechanical hand tool dexterity test.

1. SHAD

Performance tests during SHAD I and II were given before the excursion at saturation depth and during excursions. There was no difference in tracking or arithmetic performance between pre-dive and the SHAD I and II saturation environments. Tracking showed a consistent decrement during excursions in SHAD I compared to scores during saturation, and the same thing was seen in SHAD II but less distinctly. Arithmetic performance was reduced during excursions in SHAD I but not in II. The scatter of the data points was greater in II (Moeller, 1974*; 1976*).

The SHAD III 8-hour exposures to 100 fsw disclosed no decrement in performance compared to the scores at storage depth at 50 fsw. Tests were done at both the beginning and end of each excursion.

2. Nisat I

More performance tests were done in Nisat I than in the other dives.

Tracking and 5-choice reaction time performance were appreciably down during the first tests given about one hour after reaching bottom (Moeller, 1975*). The next testing 6 hours later, after two of the divers were sick, resulted in equally low scores. The oxygen was then raised, and there was a little improvement 2 hours later. By the next afternoon the scores were almost back to predive levels. There was a secondary slump on Dive Day 4, then a gradual climb to pre-dive levels by Day 5 and continued slight improvements thereafter which lasted through decompression and into the post-dive period. The latency of the reaction time tests showed essentially no change throughout the experimental period.

The cognitive tests of short term memory, addition, multiplication and digit span were slightly and somewhat sporadically affected by Nisat I.

An additional test, time estimation, was given at 2200 and 0140 after going to pressure and one or two times daily thereafter. During this test the diver was asked to estimate 10, 4, 20, 30, and 60 seconds without reference to a clock. Results of the test given 6 hours after pressurization showed marked elevations (30-50%) in the estimates (i.e., overestimations) of JB and NT, the divers most affected by the nausea, and temporary slight elevations or an occasional underestimation by RJ. This tendency did not change in the next trial at 0200, 11 hr after reaching 198 fsw, but improvements were seen the next day. Large overestimations were again seen on Day 4 by JB, moderate excesses by RJ.

An additional performance test, sequential reaction time (IV.D.3 and Weybrew, 1978*), showed a typical increase (c. 20%) in response time during the first 2 days, gradually returning to pre-dive baseline by Dive Day 3 only to go up again on Day 4. Errors on this test fluctuated about the same mean of about 10% above pre-dive throughout the dive, and showed no trends except a slight additional increase on going to pressure.

Mechanical aptitudes as demonstrated by the Bennett Hand Tool Dexterity Test (Bennett and Fear, 1943) showed only a small decrement (<5%) on going to pressure but showed a peak time (slower performance) of 27% over pre-dive on Day 4.

A visual reaction time test was performed along with the visual evoked responses on Dive Days 1, 3, 5, and 7 (McKay et al, 1977*). The diver pressed a telegraph key connected to a timer in response to a light. The mean scores for visual reaction time showed the same decrement on Days 1 and 3, then a steady linear climb in performance. In this case the curve intersected predive values at about the end of decompression.

Summarizing the performance data, there was an expected decrement not unlike that caused by air at 250 fsw (the equivalent PN_2) during the first day, which showed some recovery by the second day and returned to baseline (pre-dive) levels in most cases by the 3rd. Then several parameters showed

a drop in performance again on Day 4 or 5. Most were at baseline or better by the beginning of decompression, no doubt a combination of learning and adjustment or adaptation.

It is tempting to try to associate the decrements seen on Day 4 with the decision to switch to decaffeinated coffee on the evening of Day 3. For a person accustomed to many cups of coffee a day to be exposed to this narcotic atmosphere (there were many complaints of drowsiness and a tendency to sleep if not stimulated) and then have coffee withdrawn, it is reasonable to expect some additional drowsiness and possibly an accompanying performance decrement. However, Diver NT was not a coffee drinker so his performance on Days 4 and 5 should not have been affected by this change in routine. His tracking score showed the biggest drop of the three on Day 5, and his addition and multiplication scores were down uniquely on Day 4; his sequential reaction time and associated errors showed increases on Days 2 and 3 but were good on Day 4, and his nut and bolt test improved on Day 4, even more on 5; on the other hand his time estimation was the best of the three on Days 4 and 5. This analysis does not disprove the "coffee theory," but is not consistent with it. Incidentally, the exercise periods which were so stressful to NT were on Days 6 and 7.

3. Nişat/He II and III

Performance was not a major objective of these dives, but the effect of the isobaric switch of inert gas from nitrogen to helium in a depth range where nitrogen narcosis is presumed to be minimal was of interest. In Nisat/He II the tracking score in one of three divers dropped slightly in the first test at pressure (tests were performed the day after compression). There was no change in tracking after the switch. Reaction time score (number correct) dropped in all three at 66 fsw, and again there was no noticeable effect of the switch. Addition scores showed no deviations from a slightly improving learning curve, except in one diver with quite variable results on that test throughout the experiment.

Tracking in Nisat/He III showed a slight steady rise in 2 subjects, and level performance in the other (TG) except for one low point during the treatment phase at 129 fsw. Reaction time and addition also showed no deviations. In this dive the first human factors test following the gas switch was 2 days later.

There was no noticeable improvement in performance tests on switching to helium, perhaps because the decrement due to nitrogen at 66 and 99 fsw was slight to begin with, and since there was a long delay before measurement.

4. Adaptation

One currently fashionable question in the study of diving-induced narcosis concerns adaptation. "Adaptation" as we visualize the use of the term could manifest itself in 3 general ways: (1) The acquisition of

tolerance during a steady state exposure such as that of Nisat I or any nitrox saturation exposure of sufficient depth; (2) an improved tolerance for narcosis resulting from repetitive dives; (3) improved tolerance on deep excursions as a result of living in a hyperbaric nitrox atmosphere, such as SHAD I and II.

The Nisat I exposure clearly resulted in some adaptation of type (1), but the reason for the secondary dip in performance on Day 4 is uncertain, nor is it clear whether that has anything to do with "adaptation." All performance parameters after a drop from the initial entry into nitrogen showed a steady improvement and returned to or passed the pre-dive baseline. Is this "adaptation?"

The second category of tolerance to narcosis resulting from repetetive dives (Moeller et al, 1981) would only be relevant here only if it could be related to successive excursions. Data on that from SHAD I and II is at best equivocal (Moeller, 1975*). This latter category is the type hypothesized from the NOAA OPS data (Schmidt et al, 1974) and if seen here would have to be in the excursions. The data do not permit that type of analysis. No pre-dive performance measurements were made on the same divers compressed directly to excursion depth; and the SHAD I and II divers were at depth for a week or more before beginning excursions.

B. Nausea and the Chouteau effect

Often in science an experiment asks as many questions as it answers. This applies particularly to Nisat I. This exposure established that nitrox saturation could be conducted "safely" at 7 atm and that decompression could be managed without problems, but it also disclosed an unexpected nausea.

Actually, the nausea should not have been unexpected. It is listed as a symptom of nitrogen (or compressed air) narcosis in some references (e.g., Adolphson and Berghage, 1974) but not in others (e.g., Bennett, 1966; Shilling et al, 1976; NOAA Diving Manual, 1979). Certain previous long-duration exposures did not mention nausea as a symptom: Predictive Studies II, 2 weeks at 100 fsw, (Elcombe and Teeter, 1973); NOAA OPS II, 6 days at 120 fsw (Hamilton et al, 1973); 50 hours at 7 and 8 bar in separate tests at DFVLR (Hartmann and Fust, 1967). In the latter experiment at 8 bar one of two divers were reported anecdotally to have had symptoms similar to those in Nisat I when PO_2 was reduced from 0.6 to 0.28 bar (Oser, 1975). Albano (1970) did not report similar symptoms during manned exposures at 7 ata respiring 0.28 atm oxygen, balance nitrogen. In 49 short man dives to 87-120 msw Adolfson and Muren (1965) had one subject vomit during bottom time at 90 msw.

Nausea is a common problem of anesthesia, and is has been seen in experiments using nitrous oxide to mimic nitrogen narcosis (Hamilton, 1973). It is caused also by other "narcotic" drugs (Resnick, 1966).

Nausea had been seen in similar circumstances in divers exercising at 7 atm. During Project Hydrox II, Edel (1974) exposed 4 divers to 7 atm using an oxygen partial pressure of 0.21 atm, and either hydrogen, helium, or nitrogen as the diluent gas.

During the first of the nitrogen based dives, two of four divers (ME and PG) became dizzy and nauseated at depth after 2 hours, with exercise, and one of these (ME) subsequently vomited. Both of these divers had consumed a hearty breakfast about 5 hrs prior to the dive, the other two did not. After elevation of the chamber oxygen partial pressure, the symptoms were no longer evident. A re-exposure of these same subjects in the absence of a hearty breakfast resulted in mild nausea in PG only. Edel knew to raise the oxygen for this subject because of a previous experience, as yet unpublished (Edel, 1975).

Prior to Project Tektite, Edel completed a series of experimental dives designed to assist in the derivation of the excursion dive regimen (Edel, 1971). During these dives, human subjects were exposed to 4 atm abs total pressure breathing 0.20 atm oxygen, balance nitrogen. One of these subjects experienced signficant difficulties during this exposure. After fifteen hours under the indicated conditions and after a heavy meal, the subject could not be awakened from a deep sleep, and his companion diver observed a cyanotic coloration of his face and fingernails and abnormally slow respiration. The symptomatic diver was given compressed air by mask (PO_2 = 0.85 atm) leading to his arousal, establishment of a normal respiratory pattern, and disappearance of discoloration. Arousal took several minutes. After 10 min on air, the subject seemed normal and was returned to breathing chamber gas. Within ten mintues the subject again became unconscious and the prior symptoms returned. Again compressed air for ten minutes returned the diver to apparent normalcy. The chamber oxygen partial pressure was increased to 0.28 atm and the diver returned to this breathing medium. The symptoms did not reappear over the ensuing seventeen hours of the dive. During evaluations completed some weeks after the dive, the subject would fall asleep while respiring 0.15 atm oxygen, balance helium, at surface pressure and gradually awaken when respiring room air. This evaluation demonstrated the subject's apparent unusual sensitivity to decreased oxygen levels and may explain why the other two divers were unaffected under identical dive conditions. The sensitive diver was henceforth referred to as "Canary."

The phenomenon observed in Edel's subjects and in Nisat I may be manifestations of the "Chouteau effect." Chouteau observed that goats at high pressure and gas density (in the range of 50 atm abs) showed abnormal behavior when oxygen was at a normal partial pressure (Chouteau et al, 1967). When the oxygen was raised the animals returned to essentially normal condition.

The oxygen dependent behavioral abnormalities followed the general trend of initial lethargy and diminished alimentary activity, incontinence, proceeding to uncoordinated movement with a subsequent paresis followed by loss of consciousness and ultimately death (Chouteau, 1971). In other experiments by Chouteau (1969), when the oxygen partial pressure was maintained at 0.20 atm in a nitrogen environment, the animals behavioral abnormalities were not observed at 5.8 atm or 7.8 atm, but were observed at 10.7 atm.

If the Chouteau effect is real it is probably a matter of relative hypoxia. Nausea is not a classical symptom of acute hypoxia, but is seen in longer exposures leading to "mountain sickness" (Weihe, 1964; Porter and Knight, 1971). It is also characteristic of the chronic hypoxia due to carbon monoxide poisoning. However, the syndrome seen in Nisat I resembles hypoxia only superficially, and it alone is not likely to be full story.

What seems to make sense is a combination of factors, involving both higher nitrogen and deficiency of oxygen. The divers are able to cope with this degree of chronic narcosis as long as everything else is all right. The oxygen deficiency (presuming it exists) lowers the threshold for the narcotic effects to show up.

There are problems with the oxygen, or more properly hypoxia, theory. First, the response to raising the oxygen level took some time to show up (minutes to several hours, depending on the parameter), and was not complete. Also, the evidence is presumptive that it was the oxygen that caused all the observed improvements; some of them were timed appropriatedly for this explanation, some were not.

Frankenhauser et al (1963) observed that oxygen seemed to increase the acute effects of nitrogen narcosis. These effects, however, were not seen at the borderline of hypoxia presumed to be the situation in Nisat, but were at adequate or hyperoxic levels.

Even though performance test scores returned to normal after a few days the symptoms of narcosis persisted for about 5 days. On Dive Day 5 the divers still complained of constant drowsiness and desire to sleep and were observed to have slightly slurred speech, slow reactions, and poor time perception, and they frequently burst out into fits of laughter. Nausea was still occasionally present. After the O_2 was raised most complaints were about gas density and "heavy breathing" or the clammy feeling. On Day 10 after decompression had proceeded to 130 fsw, one diver complained that "things were not funny anymore."

Because of the profound vagal effects on the heart due to the nausea it is not possible to deduce whether hypoxia played a role in the cardiac disturbances seen early in the dive (Bellet, 1963). Diver RJ, who had less nausea, had no EKG changes except bradycardia.

Focussing on the nausea itself leads to still another hypothesis. The nausea was described as being like seasickness. It waxed and waned in its intensity, and the divers had a good appetite in the midst of it. This brings to mind the audiovestibular sickness seen in too rapid decompressions from deep heliox dives (Buhlmann and Gehring, 1976) or more specifically the isobaric counterdiffusion sickness experienced by divers in Predictive Studies III (see Lambertsen, 1976). Consider also that these divers suffered from joint pains for most of the dive period, pains similar to those of hyperbaric arthralgia (Bradley and Vorosmarti, 1974). Hyperbaric arthralgia in helium is seen as being due to "rapid" compression.

Perhaps this seasickness-like nausea could be compared to that due to CDS. Counterdiffusion sickness was originally thought to be due to gas osmosis (Blenkarn et al, 1971). In Nisat I there were a lot of molecules of a soluble inert gas, but no second inert gas to cause classical counterdiffusion problems. It is possible that this gas acted on the vestibular system in a way similar to the other gas-related problems of the inner ear, vestibular decompression sickness and counterdiffusion sickness, but was caused by an overabundance of soluble inert gas acting osmotically (Hills, 1972). Unfortunately, ENG data were not taken on this dive.

The nausea was not typical of influenza, as the divers were really not sick enough; furthermore, with the observed timing this would have been a remarkable coincidence. The possibility of a contaminant--say for example in the nitrogen--cannot be ruled out, but the gas was analyzed for known contaminants and anything else would have been spotted unless it had been present in extremely small concentrations.

VI.
DECOMPRESSION

Several categories are represented in this chapter. Three SHAD dives involved excursions. Each of the dives involved a saturation decompression, and these were of several types. The calculation methods, treatments, and means of assessing decompression success are covered, as well as the results of the decompressions. Also included are the results of switching the background inert gas.

Results of SHAD I and II confirmed the NOAA OPS concept for short descending excursions, but some ascending excursions were found to be less innocuous than had been expected. Five nitrox saturation decompressions were performed (for a total of 12 man-dives), one of them from unusually deep, and they led to 3 cases of decompression sickness. Two heliox saturations were uneventful.

The counterdiffusion gas switch resulted in an injury resembling decompression sickness, thus demonstrating potential problems with the use of this transition, and it provided some firm data to help in dealing with it. Doppler monitoring showed some good correlations with DCS (decompression sickness) symptoms, but did not detect bubbles following the gas switch. The four cases were successfully treated.

A. Computation methods and profile development

All 7 of the SHAD-Nisat dives involved unusual or experimental decompressions. A historical perspective is given in Chapter I. Acquiring the profiles and procedures is a fundamental and often pivotal step in carrying out this type of experiment; this section covers how the various profiles were devised.

The calculations for SHAD I were performed for NSMRL at the Union Carbide laboratory in Tarrytown by David J. Kenyon assisted by Mark Freitag. A complete SHAD II profile was calculated there also; it was verified by a computer run at NSMRL. The SHAD III profile was calculated entirely at NSMRL, where the same ascent constraints were used and essentially the same calculation procedures. This work was done by C.A. Harvey, Ruport Hester and George Moeller, and it drew on previous NSMRL experience in this technology (Moeller, 1966; Robertson and Moeller, 1968).

1. SHAD I and II excursions

The specific method used for making the computations and its derivation is covered in detail in the NOAA OPS report (Hamilton et al, 1973). Basically it consists of bookkeeping, keeping track of the gas taken up and released from a dozen or so "tissue" compartments, each characterized by its time constant (expressed as a half time). The gas loading in each compartment is compared with a matrix of empirically determined ascent-limiting m values (m for maximum), one for each

compartment and each depth. During ascent if the gas in all the compartments is less than their respective m values then it is considered safe to ascend. If not, then the diver has to wait at a stop until the gas loading in the tissues -- following an exponential decay based on the differential gas partial pressures in and out of the compartments -- drops below the m values. He can then ascend to the next stop. It is possible to make the calculation evaluate a series of slow linear ascents as well as staged stops. The matrix of m values used by Kenyon is identified as 32/02, or the NOAA matrix, and it is used with a calculation model having 11 compartments ranging from 5 to 480 minutes half time.

The SHAD I excursions were planned from the start as a single continuous pressure and gas profile covering the 29+ days of the dive. Other constraints included maximum compression and decompression rates of 60 and 30 fsw* /min, respectively. All excursions were to be no-stop or "no-decompression" dives, ranging between 6 and 60 minutes of bottom time, with the divers breathing air throughout. The excursion plan, which had to tolerate +/- 15 minutes variance in starting time, is shown in Table VI-1. The depths and excursion times were predetermined, and the bottom times were calculated as a function of the depth and the current gas loading.

The dives were distributed about the day in order to evaluate the possible effects of circadian rhythms. The excursions shown in Table VI-1 were the ones actually performed; the times given were the maximum allowed, the actual times excursed are shown in parentheses. Some of the excursions were moved after the calculation had been made, and several were included in the calculation that were not performed. These changes were made in a conservative way with respect to the effect of the previous excursion -- i.e., an ascending excursion might allow a closely following descending excursion to have a longer bottom time, and vice versa. The starting time tolerance and ability to shift excursions around was justified on the basis of additional calculations following different excursion patterns. The entire series was calculated conservatively with respect to the oxygen in air, considering air as 17% oxygen. Some stops could have been longer than the times given; the excursions to 75 fsw and 15 fsw were unlimited, and to 100 fsw about 600-630 minutes could have been allowed, depending on the divers' recent history.

SHAD II used the same restrictions for excursions, the only difference being the saturation depth of 60 fsw instead of 50. The SHAD II times are shown in Table VI-2. Three additional 150 fsw excursions had been included but were dropped.

2. SHAD III excursions

Calculations of the same type as done for SHAD I and II showed that the 6 desired 8-hour excursions (480 min) were all allowable. However, for added conservatism the ascent rates back to the habitat depth from 100 fsw were adjusted to require 25, 20, 15, 10, and 5 minutes to return from the first 5 excursions.

---

*fsw = feet of sea water, defined as 1/33 of a Standard Atmosphere

Table VI-1. SHAD I Excursions

Key: Depth-in-fsw / Bottom-time-in-minutes (Actual-time)

Dive day	Clock time 1000	1500	2300
9	200/18(18)		
10			
11			
12	235/6(5.9)		
13			
14	235/6(5.9)		
15			
16		235/6/(6.0)	
17	200/18(18)		
18			
19		200/18(18.7)	
20			200/18(18)
21	150/43(43)		
22	5/32(31)		
23		150/43(43)	
24			
25		15/60(57)	150/49(49)
26	150/43(43)	75/60(60)	
27	100/60(59)		15/60(55)
28	5/32(31)	200/18(18)	

Table VI-2. SHAD II Excursions

Key: Depth-in-fsw / Time-in-minutes (Actual-time)

Dive day	Clock time 1000	1500	2300
8	100/60(60)		
9			
10			
11			
12	100/60(60)		
13			
14		100/60(60)	
15			
16	150/60(60)		
17		200/20(20)	
18			
19	250/6(6)		
20	05/23(22)		
21			
22	15/37(32)	200/20(20)	
23	150/60(60)	100/60(60)	
24	100/60(60)	15/32(32)	
25	05/23(22)		200/20(20)

3. Nitrox saturation decompressions, SHAD and Nisat I

The Pre-SHAD profile was calculated to be the fastest saturation ascent that could be run using the NOAA decompression model and matrix, with a limiting half time of 480 minutes. It is shown in Table VI-3. For convenience ascent "rates" are expressed in minutes per foot, which is actually the inverse of rate.

Table VI-3
Pre-SHAD Saturation Decompression

For bottom time 12:00 to 24:00 at 50 fsw:

Decompression time, min	Rate min/fsw	to, fsw	Transit time min
0	6	10	240
240	20	5	100
340	30	sfc	150

Total decompression time 490 min = 8:10

For bottom time 24:00 to total saturation (this one was used):

0	6	20	180
180	15	10	150
330	20	5	100
430	30	sfc	150

Total decompression time 580 min = 9:40

The SHAD I saturation decompression was made more conservative in view of DCS observed after Pre-SHAD. Again 17% oxygen was used in the calculations as a conservatism ("J-") factor. The computation was started at 35 fsw, since at that point the absolute pressure was 68 fswa and that was also the inert gas load of the divers when saturated at 50 fsw. This initial more or less unrestricted ascent is considered valid as the "oxygen window." Five fsw increments were used for rate changes. An arbitrary ascent rate of 6 fsw/min was used to 55 fsw, followed by an arbitrary 15 min/fsw until the calculated limit was reached, at 20 fsw. Thereafter the rates vere rounded from the calculated values. No holds were planned. See Table VI-4.

A SHAD II saturation decompression proposed by the Union Carbide investigators would have required 1980 min or 33 hours from 60 fsw. Instead a table was calculated at NSMRL, again based on the NOAA matrix and a limiting half time of 480 minutes. Air was breathed but 17% oxygen was assumed for the calculation. An arbitrary initial ascent rate of 10 min/fsw was chosen for the oxygen window portion of the ascent, from 60 to 45 fsw. Then as in SHAD I an arbitrary 15 min/fsw was used until a matrix

Table VI-4
SHAD I Saturation Decompression

Depth fsw	Ascent Rate min/fsw
50 to 35	6
35 to 10	15
10 to 5	33
5 to sfc	36

Total decompression time 810 min = 13:30

violation caused a rate change at 19 fsw. Also a sleep hold was arbitrarily inserted after the calculations were complete, resulting in the 27 hr 42 min table given in Table VI-5. Ascent was accomplished in 1/2 fsw stages instead of the true linear ascent.

Table VI-5
SHAD II Saturation decompression

Depth fsw	Rate min/fsw
60 to 45	10
45 to 19	15
19 to 12	33
12	Hold 8 hr
12 to 5	37
5 to sfc	36

Total decompression time 1662 min = 27:42

A slightly different approach was taken in SHAD III (Table VI-6). The oxygen window to 35 fsw was used as before at 10 min/fsw, with the same 15 min/fsw initial violation at 21 fsw. The limiting half time used for this computation was 640 minutes, hence the especially slow ascent after 21 fsw. This profile was followed only to 18 fsw.

The Nisat I decompression posed a significant problem. Here the objective was to achieve a bends-free decompression without great concern for speed. In particular it was felt that the fast "first pull" should be limited; to some extent this would be the expected result of having a low oxygen partial pressure and hence a small oxygen window. The computation was made using the 640 minute limiting half time and an oxygen partial pressure of 0.3 until the oxygen level reached 21%, then air thereafter. This profile is given in Table VI-7.

Table VI-6
SHAD III Saturation Decompression

Depth fsw	Rate min/fsw
50 to 35	10 min/fsw
35 to 21	15 min/fsw
21 to 14	54 min/fsw
14 fsw	Hold 8 hr
14 fsw to sfc	54 min/fsw

Total decompression time 1428 min = 23:48
The table was interpreted at 18 fsw due to DCS.

Table VI-7
Nisat I Saturation Decompresson

Depth fsw	Rate min/fsw
198 to 188	10
188 to 177	15
177 to 14	53
14 to sfc	53 to 62

Total decompression time 9656 min = 161 hr

4. Nisat/He II and III heliox saturation decompression

Decompression from the last two Nisat/He dives was according to the standard U.S. Navy helium-oxygen saturation decompression profile, (U.S. Navy Diving Manual). This calls for an ascent rate of 15 min/fsw (4 fsw/hr) from the bottom to 50 fsw then 20 min/fsw (3 fsw/hr) to the surface. Decompression is halted each day for sleep at 1400-1600 and 2400-0600. The decompression following recompression in Nisat/He III started at 129 fsw, but the slower rate for 100-50 fsw was used from that point.

The Nisat-He II dive began at 0900 so had one 2-hour hold at 47 fsw and a 6-hour hold at 23 fsw. Total decompression time was 28.7 hours.

The Nisat/He III dive began decompression from 129 fsw at 0600. The rate of 4 fsw/hr was used from 129 to 100 fsw as well as 100 to 50. This decompression had two 2-hour and two 6-hour holds and took 54.3 hours.

5. Treatment procedures

A comprehensive set of profiles was computed by the Union Carbide laboratory for dealing with the many possible contingencies which might possibly occur during the SHAD dives. These included aborts, decompressions with low oxygen, etc.; they are discussed in II.D.10. The

methods used for computing these tables were the same as those covered above for SHAD I and II. Because they were never tested they are not included here.

B. Methods of assessing decompression

A frustrating thing about decompression science is the difficulty of grading success; it is, basically, scored on failure, the occurrence of decompression sickness. Traditionally the success of a decompression procedure has been judged on all-or-none basis, one either gets bends or does not. Some attempts were made in SHAD-Nisat to use a more sophisticated means of evaluation, Doppler ultrasonic bubble detection; some correlation was observed between bubble score and decompression stress. Also, platelet counts and serum enzyme levels were examined for changes that might be related to decompression, but none were found.

1. Decompression sickness

The ultimate assessment of decompression is whether or not the diver gets symptoms of DCS. The symptoms can be further broken down into three general categories: skin itching, pain-only joint bends, and neurological involvement. Additional breakdowns are the intensity of the symptoms and the time and frequency of occurrence. Skin, pain-only and neurological DCS are in the order of increasing severity, and concomitantly decreasing success in assessing the decompression procedure, or the "table" as it is usually called.

Only itching and pain-only decompression sickness were observed during the SHAD and Nisat dives.

2. Doppler bubble detection

It has been realized for some time that bubbles could be present in otherwise asymptomatic divers (Behnke, 1951), and in recent years circulating bubbles have been detected on a routine basis using ultrasonic equipment based on the Doppler shift (Spencer and Campbell, 1968; Spencer and Clark, 1972; Powell and Spencer, 1980; Pilmanis, 1976). This method works by means of a pair of crystals (a transmitter and a receiver) activated at ultrasonic frequencies and equipped with electronic circuitry to cause them to respond only to moving objects. The crystals are focussed such that when properly placed on the chest (on the precordium, hence the term "precordial") they can pick up signals from the pulmonary artery. Bubbles in the venous system are detected as blood passes from the right heart to the lungs. A bubble or other venous "embolus" is heard as a "chirp" or sharp, high-pitched sound that stands out against the background hissing of the blood or heart valve sounds. The bubbles detected by this method are thought to be 5-10 microns in diameter or larger. They are often referred to as VGE, venous gas emboli.

A Spencer ultrasonic Doppler flowmeter was used in SHAD and Nisat (Model 1027, Institute of Environmental Medicine and Physiology, Seattle, WA). It was operated at 5 mHz. During the SHAD dives the location of the probe was adjusted by the divers according the sounds heard by the topside investigators. The divers practiced placement of the probe before the dive, and its proper location was marked on the chest. The divers were aware of the bubble sounds as they were being recorded.

The Doppler bubble counts were estimated by the topside investigator at the time, according to an arbitrary system on a scale of 1 to 10, expressed in this report as "2/10," for example. (See Table VI-9.) These are not the familiar Spencer "bubble grades" (Spencer, 1977); we use the term "score" instead of "grade" to call attention to the difference.

Table VI-8
Doppler Scoring System

0 - "silent doppler" (none other than normal heart sounds).
1 - "isolated doppler sounds" (up to 1 per 15 sec).
2 - "occasional sounds" (1 per 15 sec to 1 per 5 sec).
3 - "scattered sounds" (1 per 5 sec to 1 per 2 sec).
4 - "frequent sounds" (1 per 2 sec to 2 per sec).
5 - "rapid sounds" (2 per sec to 5 per sec).
6 - "continuous sounds" (individually distinguishable, but too rapid to count).
7 - "continuous sounds with scattered bursts of score 9-10."
8 - "continuous sounds with frequent bursts of score 9-10."
9 - "doppler sounds sufficient to nearly obscure heart sounds."
10 - "doppler sounds sufficient to obscure heart sounds."

The plan for Doppler monitoring called for it to be performed during and after all excursion ascents, beginning on the way up during upward excursions and during the return to habitat depth after descending excursions. Monitoring was to be continuous during ascending excursions, alternating between the divers and until 5 minutes after return to habitat depth. After both ascending and descending excursions monitoring was to be carried out every 15 minutes for the next hour. This schedule was followed reasonably closely throughout the program; it was modified if bubbles were heard, and monitoring was continuing until the subject was clear of bubble sounds.

During monitoring a limb movement routine was used in an attempt to locate the source of the bubbles more precisely. Each arm was independently extended straight, elevated above the head, a hard "fist" made two or three times, and the arm was then lowered to a normal position. While sitting, each leg was independently extended and the knee flexed. While standing, a partial knee bend was completed. This routine often caused bubbles to be heard in a diver judged up to that point to be bubble free.

During Nisat/He II and III a set of coordinates was marked on each diver's chest to permit precise location of the probe. In the last two

dives the Doppler sounds were recorded on a strip chart recorder along with the ECG, but this did not provide an improvement in quantification of the Doppler bubble counts.

The results of Doppler ultrasonic monitoring are contained in the logs (Appendix A) and are extracted in the next sections. The logs show the time of monitoring, the duration of the monitoring periods, and the score if bubbles were detected.

3. Platelets and blood enzymes

Another means of assessing decompression effectiveness is by monitoring platelets (Elliott and Hallenbeck, 1975). It has been shown that 2 or 3 days after a stressful dive even without decompression sickness, platelet count shows a marked drop which lasts a day or two (Martin and Nichols, 1972), and the drop is much more prominent when decompression sickness has occurred (Philp and Gowdey, 1969). The exact mechanism for this thrombocytopenia has not been proven, but it could be due to a removal of the platelets by circulating bubbles, or to the release of serotonin resulting in platelet breakdown (Cockett, et al 1977). The gas-fluid interface off a bubble acts as a foreign body, and platelets have been observed to accumulate on bubbles (Philp, 1973).

Whatever the cause, we felt platelet counts might be an indicator of decompression stress, and platelet counts were performed periodically, usually every other day, throughout all dives. "Manual" counting methods were used, both in-house and by a commercial laboratory (Tuggle Laboratories, New London). Significant changes in platelets attributable to the events of the dives were not observed.

During SHAD I platelet counts for the two divers stayed within the range 225-350,000 throughout the entire compression period, except for a single value of 190,000 for WB in a sample taken after the first excursion. Values rose slightly in SW after saturation decompression began, from his typical level of about 270,000 to 350,000 and reached a high of 420,000 on the 12th postdive day. WB had a single low value of 215,000 on Day 27. Only the high value is outside the range of clinical normalcy. Platelet values for the "control divers" were relatively stable throughout, in the 200-300,000 range.

One SHAD II diver (RF) had an abnormally high platelet count throughout the experimental period, running routinely above 400,000 with some counts exceeding 500,000. No pattern could be seen in the SHAD II platelet counts.

SHAD III platelet values were stable pre-dive about an average value of 220,000, and increased in variability during the excursion phase with no change in average level. The single lowest value after the last excursion was 170,000 (PP), but 4 days after the completion of decompression RO showed a single low value of 145,000, at the low end of normal. The average trend for the group went from 220,000 at pressurization to 150,000 at the end of decompression, held low for 2 days, then began to climb to a post-dive level of 240,000 which prevailed during post-dive days 7 and 8.

Nisat I platelets were unchanged throughout the dive and post-dive period about an average of 270,000, except for a drop of a single sample to 210,000 3 days after surfacing.

Nisat/He II platelets increased in variability about an average of 240,000 on going to pressure, showed a slight increase 3 days after the gas shift and coincident with the start of decompression, then stabilized by the end of decompression at 250,000. Nisat/He III platelets showed essentially no variations about an average of about 220,000.

In the medical report of SHAD II (III.C) it is noted that changes in plasma enzymes could not be temporally correlated with stressful excursions or Doppler-detected bubble formation; these conclusions were corroborated by data from the other dives. A plot of the enzyme levels is given and the lack of changes in SHAD I is discussed in section IV.A.

Thus neither platelets nor blood enzyme measurements appeared to be sensitive enough to detect the decompression stress imposed by the SHAD/Nisat dives.

C. Results of the excursions

1. Descending excursions

The SHAD descending excursions, those to a pressure higher than that of the saturation habitat, were uniformly without symptoms of decompression sickness.

Doppler bubbles were detected in one diver after the 200 fsw excursion on SHAD I Day 28. No bubbles were reported following any of the descending excursions in SHAD II.

Bubbles were heard following all the excursions in SHAD III in at least one diver, and all 3 divers had bubbles at some time. There seems to have been no long-duration pattern to the bubble occurrence, in that they showed no tendency to get more or less plentiful on succeeding days. The highest score recorded was 4/10 after flexing muscles, but 1/10 or 2/10 were typical, and often no bubbles were heard unless provoked by muscle action. By way of comparison, an investigator locking in to 100 fsw for 76 min and following the prescribed decompression was scored 5/10.

2. Ascending excursions

Four types of ascending excursions were performed, to 5 and 15 fsw from saturation at 50 and 60 fsw. Each type was done 2 times. No itching was reported during any of the 4 SHAD I excursions "up" from 50 fsw, but bubbles were heard on the first two, on ascent to 15 fsw for 31 min on Day 22 and to 5 fsw for 57 min on Day 25. On the first 5 fsw excursion 3/10 spontaneous bubbles were heard 21 min after ascent and again at 31 min. These stopped on descent, at which time both divers scored 2/10 by provoking bubbles with movement.

The SHAD II ascending excursions were more eventful. Again 4 ascents were performed, to 5 fsw on Days 20 and 25, and to 15 fsw on Days 22 and 24. The upward excursions produced signs and symptoms of "skin bends" in the form of skin rash and itching (Dennison, 1971), but in only one instance were there correlated Doppler bubble sounds. Eight minutes after reaching 5 fsw on the first upward excursion Diver GS reported slight itching over the torso, forearms and upper legs. The itching increased in intensity and by fifteen minutes into the excursion was associated with a red rash on his forearms. By eighteen minutes he had Doppler sounds of 3/10 to 4/10 which persisted to recompression back to 60 feet, during which the itching diminished. Two minutes after reaching 60 feet GS had itching only behind the knees and by five minutes there was no rash or itching, though he still had 1/10 spontaneous Doppler sounds and 2/10 induced sounds when monitored 45 minutes after reaching 60 feet. By 90 minutes following the excursion no further Doppler bubbles were heard. (Some Doppler scores on this dive were taken from a medical report and were not recorded in the log.)

On the first excursion (to 5 fsw) RF developed itching of forearms after fourteen minutes, increasing in intensity and becoming associated with a rash over the forearms and stomach over the next 5 minutes. No Doppler sounds were heard from RF. Just prior to recompression he reported a dull pain of his left outer ankle, an area he apparently had struck against the bunk chains the night preceding this excursion, producing brief pain, but which had not bothered him until the end of the excursion. The ankle pain, itching and rash began resolving during recompression. The ankle pain was gone upon reaching 60 feet and the rash and itching within the next 5 minutes.

The first ascent to 15 fsw caused itching in GS behind the knee and on the forehead; no bubbles were heard on this or on the second 15 fsw excursion on Day 22.

On day 25 the last ascending excursion to 5 feet for 23 minutes resulted in both subjects reporting itching and rash of similar magnitude and distribution as that experienced on the first excursion to 5 feet on day 20, but no Doppler bubble signals were evident. The ascending excursions are anaylzed further in Chapter VIII.

D. Results of the saturation decompressions

1. Pre-SHAD

Doppler monitoring was started during Pre-SHAD after 20 fsw had been passed during the ascent from 50 fsw. A few bubbles were heard between 20 and 10 fsw, more between 10 and 5 fsw. At 5 fsw both divers noted slight numbness in the knees, which were definite symptoms by 2 fsw. Bubble counts at this time reached 4/10 in GA, 1/10 in TT. At the time of surfacing both divers had pain-only DCS in both knees, but it was vague enough that treatment was not initiated immediately. GA could not put pressure on his knees without discomfort, making walking difficult. Both

divers felt better after a warm shower about 3 hours after reaching surface.

Recompression treatment with USN Table 5 was initiated 3.5 hours after surfacing. Neither diver was promptly relieved, and the treatment was converted to Table 6. (In retrospect, Table 5 would not now be considered appropriate treatment following a nitrox saturation dive.) By the end of the treatment GA was essentially free of symptoms, TT had a painful left knee. Both divers had residual tenderness the next day, and TT had difficulty walking the following day.

Diver GA had symptoms of CNS oxygen toxicity during the last part of the oxygen breathing at 30 fsw and came off oxygen 9 minutes early. Both divers had tightness in the chest and painful coughing on taking a deep breath at the end of the treatment.

One might wonder why treatment was not started for over 3 hours after the divers surfaced with symptoms. In retrospect the symptoms were definitive, but at the time there was a legitimate question in the mind of the Duty Medical Officer. Since this was an unproven table which was to be used for a sensitive and highly visible operation it was considered important to be sure that in fact the divers did have bends. Had they been treated for "vague" symptoms there would be lingering doubts that the treatment was really required. In this case by the time treatment was started it clearly was needed, for both divers. The long time it took for the symptoms to clear completely was typical of DCS which is not treated promptly.

2. SHAD I

The SHAD I saturation decompression was completed with no symptoms of DCS reported by either diver.

Diver WB was free of Doppler bubbles throughout, but JW was scored "positive" from 40 fsw to the surface, and was judged free of bubble sounds 3 hours post surfacing (Adams et al, 1978*). One handwritten note of uncertain origin indicates that JW's bubble score was 5/10 throughout this period, with an increase to 6/10 during last quarter foot of ascent; this seems unlikely to be an accurate report, in view of his total lack of symptoms and the normal scarcity of bubbles during saturation decompressions.

3. SHAD II

During the final saturation decompression the Doppler was used intermittently, the frequency of use governed by the presence or absence of suspicious sounds. No emboli were heard, however.

During this decompression, just prior to approaching the 12 foot overnight hold subject GS complained of mild left shoulder discomfort. Physical examination revealed mild joint tenderness along the anterior biceps. The subject spent a restful night and awoke the next morning with minor residual soreness. Decompression continued and was completed uneventfully.

4. SHAD III

The last excursion to 100 fsw was over at 1800 hours, and decompression from 50 fsw began the following morning at 0942. At 23 fsw during ascent low-level Doppler bubble counts could be heard in 2 of 3 divers (PP and RO) after flexing muscles. This increased to 2/10 in PP by 19 fsw after deep knee bends. At 18 fsw, despite the reduction of ascent rate to 54 min/fsw at 21 fsw, PP reported pain in the right knee. On examination this was diagnosed as pain-only DCS, and a 10 fsw recompression was ordered. The diver felt some relief right after recompression, and was put on oxygen 26 min after recompression. He was completely relieved by the end of the first 10-min oxygen cycle. He completed 4 cycles of 10 min on, 5 off, and decompression was resumed with mild residual soreness. A 6-hour hold began at 24 fsw, which was repeated the following night (8 hours this time) at 6 fsw, after which decompression to the surface was uneventful. Occasional 1/10 bubble signals were heard.

The relatively short time between the return from the last excursion and starting saturation ascent may have been responsible for this case of DCS. In reviewing the procedures it was found that the gas loading resulting from the excursion had not been accounted for, and the 640 min gas loading compartment could have had up to 2 fsw excess supersaturation at the start of the final decompression. Whether gas loading or bubble nucleation was the mechanism is anybody's guess, but DCS was also thought to have been provoked by an excursion during Tektite. There two subjects excursed to 175 fsw for 6 hours after an accelerated saturation at 100 fsw (Edel, 1971). The 3-minute return to 100 fsw was uneventful but one diver got bends at 50 fsw, during the 30-hour decompression from 100 fsw which began 16 hours later. However, the same diver later got hit at 40 fsw on the same table, this time with no excursions; the saturation decompression was later lenghtened to 49 hours (Beckman, 1971).

5. Nisat I

The relatively conservative decompression from Nisat I was quite successful. Two divers (NT and JB) had a 10-minute session of itching on body and arms at 102 fsw. No Doppler bubbles were heard during the decompression.

6. Nisat/He II and III

The effects of the gas switch are discussed in VI.D. The standard Navy heliox saturation decompression was successful and without incident on both dives. One diver in Nisat/He III (RC) reported a mild red rash on his legs at 77 fsw, but it was judged not to represent DCS.

No Doppler bubbles were heard on either decompression.

E. Results of gas switching

1. Background

The Nisat/He dives were carried out specifically to investigate the consequences of a gas shift that might be required operationally in a certain submarine rescue situation (Greene, 1974). Isobaric counterdiffusion was first observed at the Experimental Diving Unit (Blenkarn et al, 1971) and was serendipitously rediscovered during Predictive Studies III at the Institute for Environmental Medicine (IFEM), University of Pennsylvania, in 1971 (Idicula et al, 1976). Subsequent studies at IFEM disclosed that the basic mechanism of the observed lesions was a counterdiffusion phenomenon, and further described two separate situations, the "steady state" counterdiffusion that has been observed, and the "transient" counterdiffusion that results from an abrupt gas switch (Graves et al, 1973; Harvey and Lambertsen, 1978; D'Aoust et al, 1977*).

In the steady state situation the subject is surrounded by a light (highly diffusing) inert gas such as helium and is breathing a heavy (more slowly diffusing) inert gas such as nitrogen. In the transient case the subject is saturated with a heavy inert gas and his atmosphere is switched to a lighter one. In both cases the symptoms include skin lesions and itching, but in the steady state case vestibular symptoms (vertigo, nausea) also can be expected; they have not as yet been observed after a transient switch.

The transient case had been observed in goats prior to the start of Nisat/He, and there was good reason to expect it in humans (D'Aoust, 1977a: D'Aoust et al, 1979). D'Aoust found with both pigs and goats that large numbers of bubbles could be detected with relatively minor supersaturations, and that the bubbles so generated could persist for at least 48 hours (D'Aoust, 1977b; D'Aoust et al, 1979).

2. Events

The chronology of events about the gas switches in Nisat/He II and III are given in III.F and III.G, and the mechanism used to make the transfer is given in II.B.5.

To review, within an hour after the switch from nitrogen to helium as a background gas in Nisat/He II at 66 fsw the first diver began to itch. All three divers itched, but no rash was reported; symptoms were all clear within 9 hours after the switch. No Doppler-detectable bubbles were heard.

The sequence was similar but more intense in Nisat/He III. All 3 divers itched and developed rash within an hour, but rashes and itching were gone 7 hours after the switch. All 3 divers felt some joint discomfort, and by 12 hours post-switch one of the divers had clear-cut symptoms of pain only decompression sickness (DCS), or more preferably, isobaric counterdiffusion sickness (CDS). Treatment by recompression and oxygen breathing was able to resolve all symptoms by the following morning. The lesions and itching were momentarily exacerbated by the recompression, as was the knee pain; the latter is typical of many joint pain treatments.

These divers were most affected in covered areas of the body, whereas the steady-state CDS cases reported at IFEM were most intense in exposed areas. The meaning of this is not clear. When the Nisat/He III divers put their suits back on and the suits were purged with nitrogen the itching was promptly relieved.

In the case of the skin itching the divers all recovered spontaneously without recompression treatment.

The divers in Nisat/He III, though strongly affected by the switch, did not experience any symptoms of vertigo or vestibular involvement as was seen in the IFEM steady state cases.

3. Followon

Extensive analyses have been performed on the countertransport phenomenon or phenomena involved here (e.g., Hills, 1977; Steger, 1978; Tepper et al 1979; Yount, 1982), and the Nisat experience is treated in some of them.

A workshop in 1979 (Kent, 1982) pulled many of these thoughts together, and attempted as well to standardize the terminology. It appears that the group discussed and may have reached consensus on specific terminology, but it is not reflected in the preliminary workshop report. Our choice of the term "counterdiffusion sickness" is arbitrary and independent.

VII.
OXYGEN TOLERANCE

One of the most significant experimental aspects of the four SHAD dives is the exposure of the divers to elevated oxygen tensions. This chapter reviews the exposures and their effects, and looks into some attempts at quantifying them. The oxygen levels used in the Nisat dives were well below the observable toxicity limits so those dives are not considered in this chapter. Likewise CNS toxicity is not evaluated, because the level of oxygen necessary to cause central nervous system toxicity or convulsions was never reached during the SHAD exposures except briefly during the treatment following Pre-SHAD.

A. Pulmonary oxygen toxicity

1. Background

The limits of oxygen tolerance are of interest here for two specific reasons, with additional implications. First, high oxygen levels improve decompression and are necessary for treatment of decompression sickness. Next, if air is to be used for diving then the oxygen that is a fixed part of its composition has to be dealt with. Additionally, the hazards of too little oxygen are so very serious that for the greatest degree of safety in a diving operation one should use the highest level of oxygen that can be tolerated without ill effect. The oxygen toxicity interest in SHAD is focused on the obligatory oxygen fraction in air. Resulting information will be useful operationally, but is also important because a stranded submarine crew might be exposed to hyperbaric air and therefore elevated oxygen for a few days.

The effects of pulmonary oxygen toxicity are well known to most divers. These begin as a mild pain in the center of the chest (described as "carinal" or "substernal") on deep inspiration, followed by coughing and a burning sensation on inspiration; these symptoms increase in both frequency and intensity as the exposure proceeds (Clark and Fisher, 1977).

Coincidentally with the development of symptoms there is a decrease in vital capacity. While a measureable drop in vital capacity may not precede the first subjective symptoms, it is currently the best available objective measure of lung oxygen poisoning.

Additional medical details are given in II.D.8. Also mentioned there and in section I.D.1 are the convictions by diving medical authorities that lung oxygen poisoning can be stopped by lowering the inspired oxygen, and that the symptoms of pulmonary oxygen poisoning are completely reversible in the early stages (Lambertsen, 1965; Hendricks et al, 1977).

Observations on the toxic effects of oxygen exposures on the lung reveal that the effects are not linearly related to oxygen level, increasing quite rapidly with exposure to higher PO_2's, and that at some level generally regarded as 0.5 atm PO_2 an essentially indefinite exposure is possible without detrimental effects (Clark and Lambertsen, 1971). Also, the effects of oxygen exposure are markedly reduced if the exposure is periodically interrupted by periods of normoxia or lower oxygen level (Hendricks et al, 1977). The effects of oxygen exposure in the absence of inert gas are probably greater for a given PO_2, but this is not well established and is not of concern here.

2. Lung toxicity observed in SHAD

Pulmonary function tests showed no significant changes during Pre-SHAD, but both divers developed chest pain after USN Treatment Table 6. Also, diver GA in Pre-SHAD noted twitching of his lips and came off oxygen a few minutes before the scheduled end of the treatment.

No pulmonary function changes were seen during the entire course of SHAD I and SHAD II (Dougherty et al, 1978*). These exposures were at oxygen partial pressures of about 0.52 and 0.58 atm with minor extra doses on the excursions. SHAD III had a residence depth at 0.52 atm with daily 8-hr excursions to 0.84. Two of 3 divers tolerated this well, but one (DM) began to feel chest pain near the end of the first excursion. This got progressively worse, in daily cycles, and his vital capacity changes showed the same trend, with a definite circadian cycle. One of the other SHAD III divers (PP) on a relatively minor treatment for decompression sickness experienced no chest pain but showed a substantial drop in FVC after the treatment.

The observations in SHAD lead to some revisions of the meaning of the 0.5 atm PO_2 level of exposure. It is the level below which no CPTD units accumulate[2] (see VII.B), and has been considered as an upper limit for long duration exposure. In the minds of some it is the level below which there is no pulmonary damage.

Nothing in SHAD suggests that 0.5 is not a safe pulmonary oxygen toxicity limit for long duration exposures (up to one month). In fact, SHAD II gives good cause to permit raising the safe pulmonary PO_2 limit to 0.58 atm (Dougherty et al, 1978*), as long as that level is not exceeded.

However, it seems clear from the SHAD results that it is not correct to assume there are no pulmonary effects of an oxygen exposure in the range of 0.5 atm. While there are no obvious effects of such an exposure, it may predispose the diver to pulmonary oxygen toxicity (Dougherty et al, 1978*) in a subsequent treatment or other normally tolerable hyperoxic exposure. This should be considered in operational planning.

B. Other oxygen effects

1. Red blood cells

The SHAD I and II dives showed a steady and persistent decline in hemoglobin, red blood cell count and hematocrit, which made up the collective and highly interrelated indexes of blood oxygen carrying capacity. These were steadily and significantly reduced throughout both dives and for about 6 days afterwards. They were reduced but to a lesser extent in the control subjects as well, due without doubt to the daily blood sampling. The daily samples were about 28 cc, and nearly a liter of blood was taken over the 60-day course of the SHAD I experiment, and over 1200 ml in SHAD II (Murray and Jacey, 1977*).

Reticulocytes are immature red blood cells and are an indication of red blood cell forming activity. Reticulocyte levels (IV.A) were stable through the long SHAD I and II exposures but rose prominently about a week after the dives. It appears that two forces were in effect, a slow and steady exsanquination (of both divers and control subjects) and a suppression of hematopoiesis (in the divers). Murray and Jacey (1977*) point out that this latter effect is the converse of adaptation to altitude, or the equivalent of returning to sea level from residence at altitude.

It is an open question whether this observation represents a new type of oxygen toxicity in diving. It appears to be a normal physiological response no more stressful than adapting to life in Denver or Mexico City. On the other hand, it may not be favorable to expose a person who has a reduced blood oxygen-carrying capacity to the stresses of diving. The effects of long exposures to increased levels of oxygen should be monitored when they are encountered under operational conditions.

2. Fatigue and reduction of aerobic capacity

The SHAD I divers were significantly deconditioned, and the SHAD II divers were also but to a lesser extent. No objective measurements were taken to quantify this, but both subjective feelings of the divers and observation of them by others showed that it took one to two weeks for them to return essentially to normal. They had difficulty climbing stairs, walking up slight slopes, and in any activity requiring exertion. This could be related in part to the reduction in blood oxygen capacity as mentioned in the preceding section, but for two reasons this clearly does not account for all of the observed decrement. First, the control subjects showed hematological decreases, but were not deconditioned. Also, the SHAD I divers were more affected than those in SHAD II. This seems to be due to the almost total lack of exercise during SHAD I; this deficit was corrected in SHAD II. Thus it appears that only a portion of this effect can be attributed to the oxygen exposure.

3. Effect of hyperoxia on cardiac function

A portion of the drop in heart rate seen in the SHAD dives can be attributed to the increased oxygen partial pressure (Hesser et al, 1978), but most of the other deviations from normal are probably related to gas density and other factors (IV.C.1).

4. Vascular changes

The constriction of both arteries and veins in the fundus of the retina (IV.E.1) is directly attributable to oxygen.

The inhibition of salivary secretion observed in SHAD III is clearly oxygen related and is considered a manifestation of oxygen toxicity (IV.F), but the mechanism may be due to a normal vasoconstrictive response rather than to a non-reversible toxic reaction.

Numb fingertips were reported in SHAD II (III.C), but since these symptoms were not observed in conjunction with hyperoxia and were not persistent it is unlikely that they are a manifestation of this specific type of oxygen toxicity.

C. Determining doses of oxygen exposure

The degree of lung poisoning which results from oxygen exposure is a function of several variables, but particularly the partial pressure of oxygen and the duration and pattern of the exposure. There have been several attempts to develop criteria for quantifying the exposures into a "dose" in the pharmacological sense. These have largely been based on fitting a mathematical relationship to empirical data. A good example is the formula of Stelzner (cited in Hartmann and Fust, 1967), which gives residence time in oxygen, t as:

$$t = \frac{1000}{p^3}$$

where p is the oxygen partial pressure in atmospheres. This curve considers single continuous exposures at a given pressure, but makes no allowance for a series of exposures at different pressures, or for intermittent exposures. We are not attempting here to develop a new index, or even to access broadly the existing ones, but only to express the SHAD experience in their terms.

1. UPTD and CPTD

The widely accepted means for quantifying pulmonary oxygen dose is the Unit Pulmonary Toxicity Dose (UPTD) devised by Lambertsen and colleagues (Wright, 1972). The unit dose is equivalent to an exposure to a PO_2 of 1 atm for one minute, but it recognizes that the toxic effect of the PO_2

level is more pronounced at higher levels. Thus a PO_2 of 2.0 atm generates 2.5 UPTD's in 1 minute, and 0.6 atm for 1 minute is only 0.26 units. (The UPTD equations, and charts for simplified calculation are given in the Underwater Handbook, Shilling et al, 1976, as well as in the report by Wright.)

The unit doses for each minute of an oxygen exposure can be accumulated into a Cumulative Pulmonary Toxicity Dose (CPTD). (Despite their slightly different meanings the terms CPTD and UPTD are used more or less interchangeably.)

For practical application Wright's report recommends a dose of 615 units for a routine exposure; this is the dose which can be expected to result in a 2% drop in vital capacity (VC). A dose of 1425 units, equivalent to a 10% drop in VC, is recommended as the upper limit for treatment of decompression sickness.

One acknowledged defect in the CPTD concept is that the lung recovers during normal or low oxygen periods, but the calculation makes no provision for this.

2. COTi (Cumulative oxygen toxicity index)

Another index of oxygen toxicity is the COTi (Hills, 1976). This index appeared attractive to us for monitoring pulmonary oxygen exposure because it is designed to make the influence of a particular oxygen dose diminish as it "recedes into the past." Thus it should allow for the recovery which takes place during periods when oxygen exposure is below toxic levels. The original COTi was derived and validated experimentally for CNS toxicity, but Hills proposes it for pulmonary toxicity as well. We felt it might be meaningful to evaluate the COTi over the SHAD exposures.

In developing COTi Hills observed that there seems to be a physiological parameter that determines the onset of convulsions, and that it is proportional to the excess oxygen above normal and to some function of time. If this parameter stays below a certain value then toxicity is avoided. This "imminence" parameter is calculated by adding algebraically individual index values for the recent step changes in inspired oxygen level, each as a function of the time interval of its occurrence. The times are counted backward from the moment the calculation is made. The index calculation is based on the excess PO_2 and the value of a tolerance curve at the moment. Each step and its time interval are considered independently. Steps reflecting a reduction in oxygen level are subtracted.

To determine the likelihood of a toxic event from this "imminence parameter" it is compared with a predetermined value that represents the borderline of toxicity. Thus COTi does not provide a direct measure of toxicity, but rather it estimates the condition of the organism relative to a set toxicity level at a point in time. It permits the comparison of a complex oxygen exposure profile against a set of limits determined from single exposure levels and times.

The COTi is calculated from the equation:

$$COTi = \frac{(\Delta PO_2)_1}{L_1} + \frac{(\Delta PO_2)_2}{L_2} + \cdots + \frac{(\Delta PO_2)_i}{L_i}$$

where (ΔPO_2) is the change in oxygen partial pressure applying to an interval (that is, the step change from the preceding interval), and L is the limit of oxygen tolerance for an exposure duration equal to the time since the step took place (the "imminence" parameter; Hills calls this "pi"). Each term represents an exposure interval; the subscripts for the intervals get larger as time passes. Calculating the (ΔPO_2) values is straightforward; we determined an average PO_2 for each interval to be evaluated, and used the difference in PO_2 between one interval and the one before as the ΔPO_2.

Determining a proper L value required a tolerance curve. We derived one based on the USN Oxygen Limit table, using the section for exceptional exposure (USN Diving Manual, 1981, Par. 15.2.1), and graphically extrapolated out to the PO_2 level of 0.5 atm for a long exposure. This limit is based on both CNS and lung toxicity, and should be appropriate for COTi. Although Wright uses 0.5, he states (1972) specifically that this choice as the asymtote was for convenience, not to imply that this value is rigorously established. Values used were as follows:

PO_2	Tolerance time, min
2.0	30
1.9	40
1.8	60
1.7	80
1.6	100
1.5	120
1.4	180
1.3	240
1.0	600
0.8	1440
0.6	8640
0.5	43000

The 43000 minutes for a PO_2 of 0.5 atm is intended to represent a very long time and in fact is the time accumulated in SHAD I. A set of 4 polynominal expansions were used to approximate this curve with a maximum error of about 3%.

The COTi for a given moment is the sum of all the individual terms in the equation above, from the time the PO_2 first exceeded normal through all changes up to the time for which the calculation is being made. As time passes and subsequent calculations are made, each new calculation requires computing all the elements over again and again summing them algebraically for the new COTi value. The oxygen values for each time interval do not change as the earlier elements "recede into the past" with the passing of time, but the L value used to calculate the increment does change. As the

above table shows, the longer the time which an oxygen level has to be tolerated, the lower the oxygen has to be.

To evaluate COTi the calculated value is compared with 1, at which COTi level the exposure is equivalent to that of the tolerance curve. Values below 1 indicate the diver is below the toxicity level, and when they are greater than 1 the diver has exceeded the prescribed limit.

D. Oxygen doses during SHAD

Using techniques discussed in the last section we calculated CPTD and COTi for all the SHAD dives. The results of this computation for Pre-SHAD are given in Table VII-1, as an example, and the others are described below.

1. CPTD during SHAD dives

As a basis for the CPTD computation the periods between selected times in the logs were treated as "intervals," and an average PO_2 and CPTD were determined for each interval. The CPTD calculation does not count any exposure below 0.5 atm PO_2 and it tends to exaggerate PO_2 readings as they increase above 1.0 atm. Also no accounting is made of the time over which the dose accumulates. Expressed another way, there is no provision in the CPTD calculation to account for recovery that may take place during periods of low oxygen exposure.

One possible way of dealing with this, a method being used in commercial diving, is to assign a CPTD limit for a 24-hour exposure period. There are no specific literature references to support this idea, but it is consistent with the behavior of lung oxygen toxicity. In order to test this concept with the SHAD data we calculated the accumulated CPTD for the preceding 24 hours at each point (interpolating within an interval if necessary). This value as well as the accumulated total can be compared.

Table VII-1 reveals that the total dose during Pre-SHAD for Diver TT was 711 units; GA received 679. Thus the divers had very little more than the normal 615-unit dose during the whole 2 1/2 day dive, and most of this was due to the treatment. This dose was almost all accumulated in the last 24 hours of the dive.

In SHAD I (50 fsw, PO_2 = 0.52) CPTD accumulated at a rate of about 50 units per day, with some additional for certain excursions (e.g., 43 min at 150 fsw gave 54 units). This pattern did not change throughout the dive and the grand total was 1600 units.

During SHAD II (60 fsw, PO_2 = 0.58) the daily rate was about 270 units when there was no excursion; the downward excursions added 50 or so units each, making those days total 320-400 units. The amount accumulated during SHAD I, 1600 units, was reached on the 7th day of SHAD II, which had a grand total of 7900 units.

Table VII-1. Pre-SHAD oxygen doses

Oxygen-related values for selected times derived from the log for Pre-SHAD. The PO_2 values are those breathed by the diver. The 5th column shows the time of each interval used in the analysis, and the next column shows the average PO_2 during the interval, CPTD values are given for each interval, for the preceding 24 hours, and the accumulated total for the entire dive. The COTi reading for each point covers the time since the start of the dive. This table covers exposure of Diver TT; Diver GA had a total of 679 CPTD units because he stopped oxygen breathing a few minutes early.

TIME OF DAY		DEPTH FSW	PO2 ATM	RUNNING t,MIN	INT t MIN	AVERAGE INT PO2	CPTD INT	CPTD 24 HR	CPTD TOTAL	COTi
* * * * * 1973 September 10, Dive Day 1 * * * * *										
1235	2	0	0.21							
1236	3	50	0.53	1	1	0.364	0	0	0	0.069
2400	4	50	0.51	685	684	0.515	37	37	37	0.318
* * * * * 1973 September 11, Dive Day 2 * * * * *										
0954	5	50	0.52	1279	594	0.515	32	70	70	0.368
2400	6	50	0.50	2125	846	0.510	33	65	102	0.422
* * * * * 1973 September 12, Dive Day 3 * * * * *										
0848	7	50	0.51	2653	528	0.505	12	48	114	0.429
1148	8	20	0.33	2833	180	0.420	0	40	114	0.371
1418	9	10	0.26	2983	150	0.295	0	34	114	0.278
1558	10	5	0.23	3083	100	0.245	0	30	114	0.233
1822	11	0	0.21	3227	144	0.220	0	25	114	0.188
2200	12	0	0.21	3445	218	0.210	0	16	114	0.145
2202	14	60	2.82	3447	2	1.515	5	21	119	0.910
2222	15	60	2.82	3467	20	2.820	71	92	190	1.411
2227	17	60	0.58	3472	5	0.580	1	92	191	0.416
2247	19	60	2.82	3492	20	2.820	71	163	263	1.509
2252	21	60	0.58	3497	5	0.580	1	164	264	0.513
2312	23	60	2.82	3517	20	2.820	71	235	335	1.601
2317	25	60	0.58	3522	5	0.580	1	236	337	0.603
2347	27	30	1.91	3552	30	2.365	89	324	426	1.495
* * * * * 1973 September 13, Dive Day 4 * * * * *										
0002	29	30	0.40	3567	15	0.400	0	323	426	0.604
0102	31	30	1.91	3627	60	1.910	142	464	568	1.363
0117	33	30	0.40	3642	15	0.400	0	464	568	0.666
0217	35	30	1.91	3702	60	1.910	142	604	710	1.432
0218	36	0	0.21	3703	1	1.060	1	605	711	1.053

The CPTD accumulation in SHAD III is given in the paper by Adams et al (1978*). The 8-hour excursions produced about 350 units each, and the total at the end of the last excursion was 2500 units.

Adams et al pointed out that the reduction in vital capacity observed in Diver DM reached 6% on Dive Day 5, but according to Wright (1972) would be predicted to be reduced to that value by day 4. Calculations by Hester (Adams et al, 1976*) showed that by using a baseline value of 0.57 instead of 0.50 in the CPTD calculation the predicted and observed values could be made to coincide. Whether this type of adjustment to the CPTD formula would make it fit better for other exposures is not determined by this observation. The fact that DM had chest pain well before he even had the basic 615 units suggests that the calculation needs to be more conservative, rather than less.

Another approach at making CPTD more meaningful for this type of exposure was the calculation of a 24-hour total. This did not improve the situation; the daily doses were well below 615 units when all four of the SHAD lung toxicity problems occurred.

2. COTi during SHAD dives

The COTi is for Pre-SHAD shown in the last column of Table VII-1. It exceeded 1.0 during the periods of oxygen breathing and immediately afterwards. It was higher for the successive cycles of oxygen breathing at 60 fsw, went down during the ascent from 60 to 30 fsw, then rose again in the long 60 minute cycles at 30 fsw. At the end of the last oxygen cycle it was 1.4, then dropped to 1.1 after the switch back to air.

The COTi did not reach 1.0 during any of the other SHAD dives. It was typically about 0.25, rising to 0.7 or 0.8 as the result of an excursion. During SHAD III COTi was 0.74 after the first 8-hour excursion to 100 fsw (PO_2 = 0.84) and rose slightly after each succeeding excursion, reaching a high of 0.82 after the last one.

The COTi values for USN Table 6 alone without the two days of exposure to 0.52 atm oxygen are only 0.1 to 0.2 lower than those in Table VII-1, which include the prior exposure.

Thus it appears that COTi might be valid for anticipating a convulsion, but does not seem to forecast pulmonary toxicity under these circumstances. It might be more valid if used with a tolerance curve based on the individual's own experience.

VIII.
DISCUSSION AND CONCLUSIONS

The SHAD-Nisat dives contributed a great deal of data covering human exposure to the air and nitrox saturation environments. A good portion of this data collection represents phenomenological responses, and is best interpreted in context with other data on comparable exposures. However, certain operational conclusions can be drawn about the major objective areas of living in air at pressure, making excursions, decompressing from this habitation, tolerating a dense and narcotic atmosphere, and switching to a helium mixture.

The operations with their extensive monitoring and testing were significant in themselves, particularly the two month-long SHAD I and II exposures, and the shorter but more stressful SHAD III and Nisat I. Sophisticated procedures for selecting the divers from a large "pool" had to give way to more traditional procedures using available resources, but it was learned that a good selection method is to go for personality profiles similar to the diver population, to match interest areas and aptitudes within a crew, to screen out neurotic trends and emotional problems, and to take advantage of as much diving experience as is available.

There were a wide variety of changes in biochemical, behavioral, neurological, and physiological indexes, but except for those due to oxygen and decompression/counterdiffusion none seem portentous, and nothing encountered within the scope of SHAD-Nisat seems at all likely to cause permanent damage.

A. Effects of long exposures to elevated oxygen

Two operationally-relevant situations were tested in SHAD, the long habitation in hyperbaric air and the effects of excursions on a diver so conditioned.

1. Safe habitation depth for air

SHAD I and II provided data from exposures almost a month in duration to air having an oxygen partial pressure of 0.52 and 0.58 atm. There were no pulmonary symptoms observed in either dive, and these exposures can be regarded as good evidence that these levels are operationally tolerable, with two provisos.

First, in both SHAD I and II there was a significant drop in factors related to blood oxygen carrying capacity (red cells, hemoglobin, and hematocrit). A large part of this drop was due to the extensive blood sampling, but the evidence suggests strongly that reduced red cell production was also a factor. The data obtained in SHAD do not shed any

light on whether this is a deleterious change. The changes seen, after accounting for the exsanguination, are less than seen in altitude adaptation and therefore are probably a normal and totally benign adaptation. One matter to be concerned about is letting the blood oxygen carrying capacity drop to a level low enough to put the diver at extra risk if he is exposed to a situation of marginal hypoxia. This will have to be evaluated in light of the operation itself; relatively simple finger-prick blood sampling techniques could be used for day-to-day monitoring.

The reduction in red cells no doubt made a small contribution to the deconditioning seen after SHAD I, but the use of occasional exercise in SHAD II reduced the deconditioning to a modest level even in the face of a greater loss of red cells. Surely a diver working daily in the sea will maintain his fitness and aerobic capacity. Provision should be made for exercise of divers holding in saturation but not working.

The second reservation about long-term residence in the PO_2 range 0.5 to 0.6 is that it may reduce the divers' tolerance for the oxygen breathing required for a DCS treatment. The SHAD data do not define this clearly, but suggest that it may be the case. Both divers felt chest pain after Pre-SHAD, after only 2 days of exposure at 50 fsw. No diver after SHAD I or II was exposed to high oxygen, so the effect of these long exposures was not determined. One SHAD III diver felt no chest pain but had a prominent reduction in vital capacity after a relatively mild session of oxygen breathing (added CPTD of 100 units, compared to 600 for a Table 6). The other SHAD III diver felt chest pain early in the exposure, on the first excursion, so pre-conditioning was not a factor in his case.

The numbers are small, but when 3 out of 3 divers have symptoms following a normally innocuous amount of oxygen breathing there is a strong suggestion that it may be due to the preconditioning exposure.

Thus the SHAD data lead to the conclusion that operational use of air saturation at 50 or 60 fsw is acceptable for many days or a few weeks. Deeper than this, say 70 fsw, for more than a day or two is not recommended. The conditions at 50 fsw are slightly less stressful than at 60 fsw, and can probably be tolerated longer. In all cases where this operational technique is used it is important to recognize that standard methods for treating decompression sickness using high oxygen will be limited by pulmonary oxygen toxicity, and alternate methods (such as a long "soak" at pressure in near-normoxic conditions) should be available.

2. Long working excursions breathing air

The 8-hour excursions breathing air at 100 fsw performed in SHAD III appear to be close to the limit for this excursion pattern. It is possible that Diver DM was unusually sensitive to oxygen, but other clues suggest his sensitivity is at the high side of normal. As an example, the tolerance curve derived for making COTi calculations (VII.C.2) allows about 1100 minutes at a PO_2 of 0.84; this is based on the Navy exceptional exposure values. Further, this limit is from a "standing start," with no preconditioning. Thus DM's sensitivity places him only slightly below the calculated limit (using those criteria) at the point where he showed signs of toxicity. Actual work experience in air at 100 fsw for 8-hour

uninterrupted periods is scarce if not totally unavailable, due to the decompression obligation which would normally follow this exposure if working from the surface.

The results support that the limit for a continuous work period at 100 fsw of air and for a diver "preconditioned" at 0.52 PO_2 should not exceed 8 hours. For working deeper than this it will be necessary to use either shorter or interrupted work periods or have the divers reside at a shallower depth if in air or at a lower PO_2 than 0.52 atm. Excursions to 100 fsw are limited to 6 hours under decompression procedures in the current NOAA Diving Manual (1979).

As mentioned in the last section, it is essential that operational groups using this technique also be aware of the need for special management of decompression sickness in the oxygen-sensitized person.

B. Living in high nitrogen

The SHAD-Nisat series yielded data on two prevailing questions about nitrogen or nitrox exposures, the effects of the gas environment on performance, and its implication in operational nitrox diving.

As far as the nitrogen effects are concerned Nisat I might serve as a model for a submarine crew trapped at a pressure of 200-250 fsw. Performance of complex tasks and especially those requiring higher cognitive processes (judgement, reasoning) might be degraded, especially early in the exposure, but the crew can be expected to perform routine tasks competently. The degree of degradation is a function of both the task and the individual, but should be relatively minor for ordinary psychomotor tasks, in the range of a 10-20% decrement. The effect on complex tasks or higher thought processes was not examined quantitatively in SHAD-Nisat. If the atmosphere is air the effects of high oxygen will of course dominate the situation.

The ability to perform can be expected to improve over 2 to 4 days of exposure, returning to normal for many tasks.

Data are limited and incomplete for drawing firm conclusions, but the possibility is strongly suggested by the Nisat I experience that some crew members under the same circumstances could be expected to get sick (nausea similar to seasickness). The role of oxygen was not determined definitively, but it may be that if the PO_2 is not allowed to drop too low (below, say, 0.3 atm) there may be no nausea problem. Even at normoxic levels there is a chance that such nausea may be a rare phenomenon. The secondary drop in performance that was seen in Nisat I at 4-5 days is also not likely to be a regular occurrence.

Operationally, the 200 fsw depth studied in Nisat I seems too deep to be practical for a working saturation, unless there is some compelling reason not to use conventional methods (such as breathing heliox mixtures, or doing the work on excursions). Whether deeper excursions from this depth could be practical is an open question.

Heavy or extensive work at this depth on nitrox will require extremely efficient and low resistance breathing equipment to avoid CO_2 buildup and the ensuing problems.

The SHAD experiments shed little light on the question of whether living in nitrox saturation reduces the narcotic effects on deep excursions. Performance on the 100 fsw SHAD III excursions was essentially normal.

C. Excursion decompressions

The descending excursions in SHAD were uniformly successful, and most such excursions performed since then from nitrox saturation have been free of DCS. One attempt to extend the NOAA OPS decompression model to accommodate deep and fairly long (300 fsw/60 min) excursions with stops resulted in some DCS problems in the SCORE project (Miller et al, 1976). However, the computational model seems appropriate in the shallow range, as verified by the repeated long excursions of SHAD III. Doppler-detectable bubbles offer a possible warning, but these too were relatively limited in SHAD.

The effects of the several ascending excursions are analyzed in Table VIII-1. Here it can be seen that the results observed, based on the Doppler score and the symptoms reported, line up quite well with the severity of the exposure based on the differential pressure. Quite predictably the 55 fsw excursion distance caused the greatest symptoms and bubble scores, and the 35 fsw the least. There were not enough results to

Table VIII-1
Effects of Ascending Excursions
Showing symptoms and bubble score (out of a possible 10) for each situation

	SHAD I		SHAD II	
Exc to, fsw	5	15	5	15
Differential, fsw	45	35	55	45
1st exc, Diver 1	none 3/10	none 0/10	itching, rash 4/10	itching 0/10
Diver 2	none 2/10 (P)	none 2/10*	itching, rash, ankle pain 0/10	none 0/10
2nd exc, Diver 1	none 0/10	none 0/10	itching, rash 0/10	none 0/10
Diver 2	none 0/10	none 0/10	itching, rash 0/10	none 0/10

(P) = provoked by movement * = 15 seconds only

distinguish between the two 45 fsw differentials on the basis of the starting (or maximum ascent) depth; one caused itching (SHAD II, 60 to 15 fsw) and one gave a definitive bubble count (SHAD I, 50 to 5 fsw).

One thing seems clear, the difference between 45 and 55 fsw differential makes a big difference in symptoms, and would suggest that the line for operational safety lies in this range.

D. Saturation decompressions

Pre-SHAD showed convincingly that the 480-min half time is too fast for nitrox saturation decompression, at least when using the NOAA OPS ascent constraints.

The SHAD III decompression which resulted in a bend at 18 fsw is more difficult to assess. This table was calculated with a 640 min half time for the slowest compartment, but the diver had symptoms very soon after the slow ascent rates dictated by this half time (54 min/fsw) came into effect. It is tempting to blame bubble nucleation resulting from the last excursion 16 hours previous to the start of decompression. Another possibility is that the excursions created a gas loading that was not given enough consideration in the choice of ascent rates. It is significant that Doppler bubbles were heard during this decompression.

The Nisat I decompression which used the 640 minute compartment for ascent constraints over virtually the entire decompression was quite successful, producing neither symptoms nor Doppler bubbles. There is no way from the data obtained on this dive to assess whether the ascent was too conservative.

One conclusion that might be drawn from the Nisat/He dives is that with all its problems, switching to helium greatly improves the chances of a safe and relatively fast saturation decompression. A slow or partial shift to He may be a way of chopping a day or two off a long nitrox decompression.

Because the gas switch is likely to have generated bubbles (though no VGE were detected with the Doppler equipment), the clean results of these two saturation decompressions further emphasize the special difficulty of decompression from nitrox saturation. Switching to helium -- but using a proper means of accounting for counterdiffusion -- might be a feasible operational technique. Keller has proposed an approach (1979).

F. Consequences of gas switching

Given the gas tensions and time of occurrence of specific symptoms it is tempting to consider that traditional gas loading calculations can be used to predict how gas switching can be managed safely. Many of the factors involved and problems with this approach are covered by D'Aoust and colleagues (1977b; 1977*), but it does seem reasonable that useable procedures might be developed. The same methods used in calculating the

excursions were used successfully in a relatively unstressful commercial counterdiffusion situation (Peterson et al, 1980).

Supersaturation peaks were calculated for D'Aoust's goats (1977*), and he found that theoretical gas loading peaks occurred at roughly the duration of the N2 half time following the switch. That is, for the compartment having a 2-hr nitrogen half time the peak occurs about 2 hours after the shift.

On the basis of symptoms observed we can conclude that the peak which leads to itching is 60 minutes or less, but the limb bend may have resulted from a peak as long as 8 hours after the switch. The consequences are that gas shifts may have to be made over very long times -- many hours -- in order to spread the peak out enough to avoid such problems. Of course there is no guarantee that the gas loading has to reach a "peak" in order to cause symptoms. Possible ways of reducing this time would be to end up with a tri-mix that can be maintained for many hours, or to carry out a switch with simultaneous addition of some total pressure to overcome the excess gas loading.

IX.
BIBLIOGRAPHY

A. Publications resulting from SHAD/Nisat (References marked * in text.)

Adams, G.M. Shallow saturation dive using compressed air. U.S. Navy Medicine 63:4-6, 1974.

Adams, G.M. Shallow habitat air dive. I: Thirty days at a simulated depth of 50 feet of sea water with excursions between 5 and 235 feet. Undersea Biomed. Res. 1(1):A-30-31, 1974.

Adams, G., R. Williamson, C. Harvey, R. Murray and R. Hester. Shallow habitat air diving with excursions between 5 and 250 fswg: a review of four simulated dives. In: Underwater Physiology VI, edited by C.W. Shilling and M.W. Beckett. Bethesda, MD: Undersea Medical Society, 1978.

Bondi, K.R., D.D. Wray and J.E. Jordan. Implementing an exchange from nitrogen to helium in a large hyperbaric chamber. NSMRL Rept. 855. Groton, CT: Naval Submarine Med. Res. Lab., Sep 1977.

D'Aoust, B.G., K.H. Smith, H.T. Swanson, R. White, C.A. Harvey, W.L. Hunter, T.S. Neuman and R.F. Goad. Venous gas bubbles: production by transient, deep isobaric counterdiffusion of helium against nitrogen. Science 197:889-891, 1977.

Dougherty, J.H., Jr., R.L. Frayre, D.A. Miller and K.E. Schaefer. Pulmonary function during shallow habitat air dives (SHAD I,II,III). In: Underwater physiology VI, edited by C.W. Shilling and M.W. Beckett. Bethesda, MD: Undersea Medical Society, 1978.

Harvey, C.A. Shallow saturation hyperbaric exposures to nitrogen-oxygen environments and isobaric switches to helium-oxygen. Undersea Biomed. Res. 4(1):A15, Mar 1977.

Heyder, E., M.J. Jacey and D.V. Tappan. Biochemical studies of saturation and saturation-excursion dives breathing O_2-N_2 mixtures. Aviation Space Environ. Med. 50(1):51-59, Jan 1979.

Kinney, J.A.S., S.M. Luria, M.S. Strauss, C.L. McKay and H.M. Paulson. Shallow habitat air dive series (SHAD I and II): The effects on visual performance and physiology. NSMRL Rept. 793. Groton, CT: Naval Submarine Med. Res. Lab., Oct 1974.

McKay, C.L., M.S. Strauss, J.A.S. Kinney and S.M. Luria. Visual evoked responses, EEG's and reaction time during a normoxic saturation dive, Nisat I. Undersea Biomed. Res. 4(2):131-145, 1977.

Miller, D.A. and R.G. Esquire. Hyperoxic inhibition of parotid primary secretion in man. Undersea Biomed. Res. 3(1):A16, 1976.

Miller, J.W., G.M. Adams, P.B. Bennett, R.E. Clarke, R.W. Hamilton, Jr., D.J. Kenyon and R.I. Wicklund. Vertical excursions breathing air from nitrogen-oxygen or air saturation exposures. Rockville, MD: Natl. Oceanic Atmospheric Admin., May 1976.

Moeller, G. Human factors studies of hydrospace systems. AIAA paper No. 74-1290. New York: American Institute of Aeronautics and Astronautics, 1974.

Moeller, G. Inert gas narcosis limitations on nitrogen-oxygen diving. Undersea Biomed. Res. 4(1):A50, 1977.

Moeller, G. Nitrogen narcosis in prolonged and repetitive exposures to hyperbaric air. In: Underwater '75, Proceedings of the Fourth World Congress of Underwater Activites, Vol. II, edited by J. Adolfson. Stockholm: Almqvist & Wiksell, 1976.

Murray, R.D. and M.J. Jacey. Shallow habitat Air Dives I and II: Human hematologic responses to compressed air saturation diving. Aviat. Space Environ. Med. 48:1012-1017, Nov 1977.

Murray, R.D., G.M. Adams and P.A. Miles. Shallow habitat air dive I: Effect on selected hematological constituents. Undersea Biomed. Res. 1(1):A31, 1974.

Schaefer, K.E., J.H. Dougherty, J.M. Wilson, R.L. Frayre, and D.R. Knight. CO_2 retention and ECG changes in exercise during prolonged hyperbaric N_2-O_2 breathing. Submitted to J. Appl. Physiol. press)

Schaefer, K.E., J. Wilson, J.H. Dougherty and A. Messier. Physiological limits in shallow water diving using N_2-O_2 mixtures. Undersea Biomed. Res. 4(1):A49, Mar 1977.

Stewart, S.D. Shallow habitat air diving I: Selected serum protein responses. Undersea Biomed. Res. 1(1):A31-32, 1974.

Tappan, D.V., M.J. Jacey and E. Heyder. Biochemical responses to shallow nitrogen-oxygen diving. Undersea Biomed. Res. 4(1):A16-17, Mar 1977.

Weybrew, B.B. Diver adaptability during a nitrox saturation dive at 7 ATA. Undersea Biomed. Res. 5(3):159-173, 1978.

Weybrew, B.B. Shallow habitat air dive (SHAD I): Psychological screening of divers as subjects for long duration saturation experimentation. NSMRL Rept. 776. Groton, CT: Naval Submarine Med. Res. Lab., May 1974.

Wilson, J.M., P. Kligfield, K.E. Schaefer, and C.A. Harvey. Human ECG changes in rest during prolonged exposure to increased pressure breathing nitrogen-oxygen mixtures. Fed. Proc. 35:237, Mar 1976.

Wilson, J.M., P. Kligfield, G.M. Adams, C. Harvey and K.E. Schaefer. Human ECG changes during prolonged exposures breathing N_2-O_2 mixtures. J. Appl. Physiol. 42:614-623, Apr 1977.

B. References (* in text indicates SHAD/Nisat publications, IX.A.)

Adolfson, J. and A. Muren. Air breathing at 13 atmospheres. Psychological and physiological observations. Forsvarsmedicin 1:31-37, 1965.

Adolfson, J. and T. Berghage. Perception and performance under water. New York: John Wiley & Sons, 1974.

Albano, G. Principles and observations on the physiology of the scuba diver. Arlington, VA: Office of Naval Res., 1970.

Bardin, H. Penndec: A computer oriented language for recording decompression experiments. Philadelphia: Institute for Environmental Medicine, University of Pennsylvania, Sept 1973.

Bazett, H.C. An analysis of the time relation of electrocardiograms. Am. Heart J. 7:353-370, 1920.

Beckman, E.L. Medical support program. In: Tektite II, Scientists in the Sea, edited by J.W. Miller, J.G. VanDerwalker and R.A. Waller. Washington: U.S. Department of the Interior, Aug 1971.

Beckwith, J.R. Grant's clinical electrocardiography: The spatial vector approach. New York: McGraw Hill, 1970.

Behnke, A.R. Effects of high pressures; prevention and treatment of compressed air illness. The Medical Clinics of North America, p. 1213-1237, 1942.

Behnke, A.R. Decompression sickness following exposure to high pressures. In: Decompression sickness, edited by J.F. Fulton. Philadelphia: W.B. Saunders, 1951.

Bellet, S. Clinical disorders of the heart beat. Philadelphia: Lea and Febiger, 1963.

Bennett, P.B. The aetiology of compressed air intoxication and inert gas narcosis. London: Pergamon Press, 1966.

Bennett, G.K. and R.A. Fear. Mechanical comprehension and dexerity. Personnel J. 22:12-17, 1943.

Blenkarn, G.D., C. Aquadro, B.A. Hills and H.A. Saltzman. Urticaria following the sequential breathing of various inert gases at a constant ambient pressure of 7 ata: A possible manifestation of gas-induced osmosis. Aerosp. Med. 42:141-146, 1971.

Bond, G.F. Effects of new and artificial environments of human physiology. Arch. Environ. Health 12:85-90, 1966.

Bond, G.F. New developments in high pressure living. Arch. Environ. Health 9:310-314, 1964.

Bradley, M.E. and J. Vorosmarti. Hyperbaric arthralgia during helium-oxygen dives from 100 to 850 fsw. Undersea Biomed. Res. 1:151-167, 1974.

Buhlmann, A.A. and H. Gehring. Inner ear disorders resulting from inadequate decompression in "Vertigo bends." In: Underwater physiology V, edited by C.J. Lambertsen. Bethesda, MD: FASEB, 1976.

Centro de Buceo de la Armada. Penetracion del hombre bajo el agua, Proyecto "Tonofond:" Informe preliminar de la experiencia "Tonofond-10". Cartagena, Espana: Centro de Buceo de la Armada, Jan 1976.

Chouteau, J. Respiratory gas exchange in animals during exposure to extreme ambient pressure. In: Underwater physiology IV, edited by C.J. Lambertsen. New York: Academic Press, 1971.

Chouteau, J. Saturation diving: The Conshelf experiments. In: The physiology and medicine of diving and compressed air work, edited by P.B. Bennett and D.H. Elliott. London: Bailliere Tindall and Cassell, 1969.

Chouteau, J., J.Y. Cousteau and J. Alinat. Manifestations hypoxiques lors de la respiration sous pression de melanges respiratoires normoxiques. J. Physiologie 59:376-377, 1967.

CIRIA UEG. Oxy-helium saturation diving tables. Rept. UR11. London: CIRIA Underwater Engineering Group, 1978.

Clark, J.M. and A.B. Fisher. Oxygen toxicity and extension of tolerance in oxygen toxicity. In: Hyperbaric Oxygen Toxicity, edited by J.C. Davis and T.K. Hunt. Bethesda, MD: Undersea Medical Society, 1977.

Clark, J.M. and C.J. Lambertsen. Pulmonary oxygen toxicity: a review. Pharmocological Reviews. 23(2):37-133, 1971.

Cockett, A.T.K., S.M. Pauley, D.N. Zehl, A. Pilmanis and W.S. Cockett. Blood and lipid alterations in early decompression sickness. In: Early diagnosis of decompression sickness, edited by R. Pearson. UMS Rept. 7-30-77. Bethesda, MD: Undersea Medical Society, 1977.

Cousteau, J.Y. At home in the sea. National Geographic 125:465-507, 1964.

D'Aoust, B.G. Transient isobaric countertransport of heliox against nitrox: Bubble production and species differences at 132 fsw. Undersea Biomed. Res. 4(1):A14, Mar 1977a.

D'Aoust, B.G. Theoretical and practical considerations of concepts of ascent criteria. In: Early diagnosis of decompression sickness, edited by R. Pearson, p. 211. Rept. UMS: 7-30-77. Bethesda, MD: Undersea Medical Society, 1977b.

D'Aoust, B.G., K.H. Smith, H.T. Swanson, R. White, L. Stayton and J. Moore. Prolonged bubble production by transient isobaric counterequilibration of helium against nitrogen. Undersea Biomed. Res. 6(2):109-125, 1979.

Dembert, M.L., L.W. Mooney, A.M. Ostfeld and P.G. Lacroix. Multiphasic health profiles in 200 U.S. Navy divers. Undersea Biomed. Res., in press.

Dennison, W.L. A review of the pathogenesis of skin bends. NSRML Rept. 660. Groton, CT: Naval Submarine Medical Research Lab., Apr 1971.

Dorr, V.A. and H.R. Schreiner. Region of non-combustion, flammability limits of hydrogen-oxygen mixtures, full scale combustion and extinguishing tests and screening of flame-resistant materials. Second Summary Report, Contract N00014-66-C-0149. Tonawanda, NY: Ocean Systems, Inc., May 1969.

Dougherty, J.H., Jr., D.J. Styer, R.G. Eckenhoff and W.L. Hunter, Jr. The effects of hyperbaric and hyperoxic conditions on pulmonary function during prolonged hyperbaric chamber air saturation dives. Undersea Biomed. Res. 8(1, Suppl.):29-30, 1981.

Eckenhoff, R.G., W.L. Hunter, Jr., J.W. Parker, J.H. Dougherty, Jr., G.O. Moeller, D.V. Tappan, and J.E. Jordan. Progress of an air saturation dive series: Airsat. Undersea Biomed. Res. 8(1, Suppl.):39-40, 1981.

Edel, P.O. Report on Project Hydrox II. Final report to the Office of Naval Research on Contract N00014-73-C-0233. Harvey, LA: Michel Lecler, Inc., Apr 1974.

Edel, P.O. Sea Space Research, Inc., Harvey, LA. Personal Communication, 1975.

Edel, P.O. Delineation of emergency surface decompression and treatment procedures for Project Tektite Aquanauts. Aerosp. Med. 42:616-621, 1971.

Edel, P.O. Experiments to determine decompression tables for the Tektite II 100 fsw mission. Report for NASA under contract 14-01-0001-1384. Houston: J & J Maine Diving, Mar 1971.

Elcombe, D.D. and J.H. Teeter. Nitrogen narcosis during a 14-day continuous exposure to 5.2% O2 in N2 at pressure equivalent to 100 fsw (4 ata). Aerosp. Med. 44(7, Sec. II):864-869, 1973.

Elliott, D.H. and J.M. Hallenbeck. The pathophysiology of decompression sickness. In: The physiology and medicine of diving and compressed air work, edited by P.B. Bennett and D.H. Elliott, p. 435-455. London: Bailliere and Tindall, 1975.

Farmer, J.O., W.G. Thomas, and M. Preslar. Human auditory responses during hyperbaric helium-oxygen exposures. Surgical Forum 22:456-458, 1971.

Ferris, S.H. and S.A. Shaffer. 35 day, 60 foot air saturation dive with rats: Effects on EEG and visually evoked cortical response. NSRML Rept. 756. Groton, CT: Naval Submarine Med. Res. Lab., Sep 1973.

Frankenhauser, M. and C.M. Hesser. Effects on psychomotor functions of different nitrogen-oxygen gas mixtures at increased ambient pressure. Acta. Physiol. Scand. 59:400-409, 1963.

Gottlieb, S.F. Effect of hyperbaric oxygen on microorganisms. Ann. Rev. Microbiol. 25:111-152, 1971.

Graves, D.J., J. Idicula, C.J. Lambertsen and J.A. Quinn. Bubble formation resulting from counterdiffusion supersaturation: A possible explanation for inert gas "urticaria" and vertigo. Physiol. Med. Biol. 18:256-264, 1973.

Greene, K.M. Theoretical considerations on the decompression of rescued submarine personnel. Laboratory report. Philadelphia: Institute for Environmental Medicine, University of Pennsylvania, 1974.

Grinevich, V.A. [Results of series of experimental studies in shore-based hypberbaric complex.] In: Podvodnyye mediko-fiziologicheskiye issledovaniya, editor in chief A.Z. Kolchinskaya. Kiev: Naukova Dumka Press, 1975.

Grinker, R. and J. Spiegel. Men under stress. New York: McGraw Hill, 1945.

Grober, Ellen. The effects of a normoxic nitrogen saturation dive on the intracellular sodium and potassium of human erythrocytes. Unpublished report, Brown University, 1975.

Hamilton, R.W. Comparative narcotic effects jn performance tests of nitrous oxide and hyperbaric nitrogen. Federation Proc. 32(3):682, 1973.

Hamilton, R.W., Jr., D.J. Kenyon, M. Freitag and H.R. Schreiner. NOAA OPS I and II: Formulation of excursion procedures for shallow undersea habitats. UCRI-731. Tarrytown, NY: Union Carbide Corp., 1973.

Hartmann, H., and H.-D. Fust. Langziet-Druchkammerversuche im Bereich von 4 bis 23 ata. Draeger-Hefte (Luebeck) 265:2-18, Oct 1966.

Hartmann, H. and H.D. Fust. Long-term pressure chamber tests in the range of 4 to 23 atm. abs. Drager Review (Luebeck) 16:2-17, 1967.

Harvey, C.A. and C.J. Lambertsen. Deep tissue isobaric inert gas exchange: Predictions during normoxic helium, neon and nitrogen breathing at 1200 fsw. In: Underwater Physiology VI, edited by C.J. Lambertsen and M.W. Beckett, p. 343-357. Bethesda, MD: Undersea Medical Soc., 1978.

Haugaard, N. Poisoning of cellular reactions by oxygen. Ann. N.Y. Acad. Sci. 177:736-744, 1965.

Heavers, B.A., A.A. Messier, R. Montanti and K.E. Schaefer. CO2 effects on acid-base balance in air saturation dives. NSMRL Rept. 742. Groton, CT: U.S. Navy Submarine Medical Research Lab., Apr 1973.

Hendricks, P.L., D.A. Hall, W.L. Hunter, Jr. and P.A. Haley. Extension of pulmonary O2 tolerance in man at 2 ata by intermittent O2 exposure. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 42(4):593-599, 1977.

Hennessy, T.R., R.G. Hanson, H.V. Hempleman and T.G. Shields. Definitive experiments to determine decompression rules for multiple air excursions from air-saturation storage. In: Undersea Biomed. Res. 8(1, Suppl.):1, Mar 1981.

Hesser, C.M., L. Fagraeus and D. Linnarson. Respiratory gases and hyperbaric bradycardia. Med. Aeronaut. Space, Med. Subaquat. Hyperbare 17(65):110- 113, 1978.

Hills, B.A. A cumulative oxygen toxicity index allowing for the regression of effects at low inspired oxygen partial pressures. Rept. 4/76. London: Royal Navy Physiological Laboratory, 1976.

Hills, B.A. Peak osmotic water displacement induced by gases in tissue. Phys. Med. Biol. 17(2):277-287, 1972.

Hills, B.A. Supersaturation by counterperfusion and diffusion of gases. J. Appl. Physiol. 42:758-760, 1977.

Houlihan, R.T. and J.R. Downs. Parotid gland response following exposure to oxygen at high pressure. Physiologist 15(3):174, 1972.

Idicula, J., D.J. Graves, J.A. Quinn and C.J. Lambertsen. Bubble formation resulting from steady counterdiffusion of two inert gases. In: Underwater physiology V, edited by C.J. Lambertsen. Bethesda, MD: FASEB, 1976.

Jacey, M.J. and D.V. Tappan. Metabolic and hematologic factors in chronic air saturation at 2.5 ata. NSMRL Rept. 781. Groton, CT: Naval Submarine Med. Res. Lab., Mar 1974.

Keller, H. Introduction of counterdiffusion effects into Neo-Haldanian decompression calculus for deep dives with multiple gas mixtures. Undersea Biomed. Res. 6(1, Suppl.):11, 1979.

Kent, M.B. editor. Isobaric inert gas counterdiffusion. Preliminary report to the Office of Naval Research under contract N00014-74-C-0319. Bethesda, MD: Undersea Medical Society, 1982.

Kindwall, E.P. Medical aspects of commercial diving and compressed air work. In: Occupational medicine, edited by Carl Zenz. Chicago: Yearbook, 1975.

Kinney, J., S.M. Luria and M.S. Strauss. Visual evoked responses and EEG's during shallow saturation diving. Aerospace Med. 45(9):1017-1025, 1974.

Kinney, J.A.S., E.J. Sweeney and A.P. Ryan. A new test of scotopic sensitivity. NSMRL Rept. 342. Groton, CT: Naval Submarine Med. Res. Lab., 1960.

Koblick, I.G. and J.W. Miller. History of Undersea Habitats. New York: Marcel Dekker (In press).

Kossman, C.E. The normal electrocardiogram. Circulation 8:920-936, 1953.

Lambertsen, C.J. Collaborative investigation of limits of human tolerance to pressurization with helium, neon and nitrogen. Simulation of density equivalent to helium-oxygen respiration at depths of 2000, 3000, 4000, and 5000 feet of sea water. In: Underwater Physiology V, edited by C.J. Lambertsen. Bethesda, MD: FASEB, 1976.

Lambertsen, C.J. Effects of oxygen at high partial pressure. In: Handbook of Physiology, Sect. 3: Respiration, Vol. II, edited by W.O. Fenn and H. Rahn. Washington: American Physiological Society, 1965.

Lambertsen, C.J. and W.B. Wright. Multiday exposure of men to high nitrogen pressure and increased airway resistance at natural inspired oxygen tension. Aerospace Med. 44(7, Section II):821-869, 1973.

Larkin, E.C., J.D. Adams, W.T. Williams and D.M. Duncan. Hematological response to hypobaric hyperoxia. Am. J. Physiol. 223:431-437, 1972.

Larkin, E.C., W.T. Williams and F. Ulvedal. Human hematologic responses to 4 hr of isobaric hyperoxic exposure (100% oxygen at 760 mmHg). J. Appl. Physiol. 24:417-421, 1973.

Larson, R.T. and W.F. Mazzone. Excursion diving from saturation exposures at depth. In: Underwater physiology III, edited by C.J. Lambertsen. Baltimore: Williams and Wilkins, 1967.

Link, E.A. Our Man-in-Sea Project. National Geographic 123:713-731, 1963.

Linmann, J.W. and R.V. Pierre. Studies on the erythropoietic effects of hyperbaric hyperoxia. Ann. N.Y. Acad. Sci. 149:25-33, 1968.

MacInnis, J.B. and G.F. Bond. Saturation diving: Man-in-Sea and Sealab. In: The physiology and medicine of diving and compressed air work, edited by P.B. Bennett and D.H. Elliott, p. 505-523. London: Bailliere Tyndall and Cassell, 1969.

Markham, T. Physiological effects of long duration habitation in hyperbaric air and artificial environments. Form DD 1498, dated 1970 Feb 5, NSMRL.

Martin, K.J. and G. Nichols. Observations on platelet changes in man, after simulated diving. Aerosp. Med. 43:827-830, 1972.

Miller, J.W., and C.J. Lambertsen. Project Tektite I: An open-sea study of prolonged exposures to a nitrogen-oxygen environment at increased ambient pressure. In: Underwater Physiology IV, edited by C.J. Lambertsen, p. 551-558. New York: Academic Press, 1971.

Miller, J.W., J.G. VanDerwalker and R.A. Waller, editors. Tektite II, Scientists in the Sea. Washington: U.S. Department of the Interior, Aug 1971.

Moeller, G. Standive, Fortran solution of decompression equations. NSMRL Rept. 465. Groton, CT: Naval Submarine Med. Res. Lab., Jan 1966.

Moeller, G., C. Chattin, W. Rogers, K. Laxar and B. Ryack. Performance effects with repeated exposure to the diving environment. J. Appl. Psych. 66(4):502-510, 1981.

Murray, R.D., S.D. Stewart, E. Heyder and G.M. Adams. Animal saturation diving at simulated depths of 50 and 60 feet: Description of operation and environment. NSMRL Rept. 772. Groton, CT: Naval Submarine Med. Res. Lab., Feb 1974.

NOAA Diving Manual. Second edition, edited by J.W. Miller. Washington: U.S. Dept. of Commerce, 1979.

Ornhagen, H. Hyperbaric bradycardia and arrhythmia: Experiments in liquid-breathing mice and sinus node preparations. Lund, Sweden: University of Lund, 1977.

Oser, H. DFVLR, Bad Godesberg, W. Germany, personal communication, 1975.

Peterson, R.E., R.W. Hamilton, and I. Curtsell. Control of counterdiffusion problems in underwater dry welding. In: International Diving Symposium '80. Gretna, LA: Assoc. of Diving Contractors, 1980.

Philp, R.B. Historical evolution of the blood-bubble interaction hypothesis in the pathogenesis of decompression sickness. In: Blood-bubble interaction in decompression sickness, edited by K.N. Ackles, p. 5-16. Toronto: DCIEM, 1973.

Philp, R.B. and C.W. Gowdey. Platelets as an etiological factor in experimental decompression sickness. J. Occp. Med. 11:257-258, 1969.

Pilmanis, A.A. Intravenous gas emboli in man after compressed air ocean diving. Final report to the Office of Naval Research under contract N00014- 67-A-0269-0026. Avalon, CA: Catalina Marine Science Center, Sep 1976.

Porter, R. and J. Knight, editors. High altitude physiology: Cardiac and respiratory aspects. London: Churchill Livingstone, 1971.

Potomac Research. Hyperbaric complex operating manual. Waterford, Ct: Potomac Research, Undated (1981).

Powell, M.R. and M.P. Spencer. The pathophysiology of decompression sickness and the effects of doppler detectable bubbles. ONR Rept. N00014-73-C-0094. Seattle, WA: Institute of Applied Physiol. and Med., Dec 1980.

Powell, M.R., G.F. Doebbler, and R.W. Hamilton. Serum enzyme level changes in pigs following decompression trauma. Aerosp. Med. 45:519-524, 1974.

Ray, C. and E. Ciampi. Underwater Guide to Marine Life. New Jersey: A.S. Barnes and Co., 1956.

Resnik, W.H. Anorexia, nausea and vomiting. In: Principles of internal medicine, edited by T.R. Harrison et al. 5th edition. New York: McGraw Hill, 1966.

Robertson, J.S. and G. Moeller. Computation of continuous decompression schedules for deep sea divers. NSMC Rept. 517. New London, CT: Brookhaven National Laboratory and Naval Submarine Medical Center, Mar 1968.

Sawyer, R.N. and J.H. Baker. The longitudinal health survey: I. Description. NSMRL Rept. 733. Groton, CT: Naval Submarine Med. Res. Lab., 1972.

Schmidt, T.C., R.W. Hamilton, Jr., G. Moeller and C.P. Chattin. Cognitive and psychomotor performance during NOAA OPS I and II. Tech. Memo. CRL-T-799. Tarrytown, NY: Union Carbide Corp., 1974.

Shilling, C.W., J.A. Hawkins and R.A. Hansen. The influence of increased barometric pressure on the pulse rate and arterial blood pressure. U.S. Navy Med. Bull. 34:39-47, 1936.

Shilling, C.W., M.F. Werts and Nancy R. Schandelmeier, editors. The Underwater Handbook: A Guide to Physiology and Performance for the Engineer. New York: Plenum Press, 1976.

Spencer, M.P. Doppler VGE and history of ultrasound detection. In: Early diagnosis of decompression sickness, edited by R. Pearson. UMS Rept. 7-30-77. Bethesda, MD: Undersea Medical Society, 1977.

Spencer, M.P. and S.D. Campbell. Development of bubbles in venous and arterial blood during hyperbaric decompression. Bull. of the Mason Clinic 22(1):26-32, 1968.

Spencer, M.P. and J.F. Clark. Precordial monitoring of pulmonary gas embolism and decompression bubbles. Aerosp. Med. 43:762, 1972.

Steger, J.W. The isobaric inert gas syndrome: A review and analysis. Unpublished thesis. Groton, CT: Naval Undersea Medical Institute, 1978.

Tansey, W.A. The longitudinal health study: A multiphasic medical surveillance program for USN submarine and diving personnel. NSMRL Rept. 786. Groton, CT: Naval Submarine Med. Res. Lab., 1974.

Tepper, R.S., E.N. Lightfoot, A. Baz and E.H. Lanphier. Inert gas transport in microcirculation: Risk of isobaric supersaturation. J. Appl. Physiol. 46(6):1157-1163, 1979.

U.S. Navy. Manual of the Medical Department. Washington: Dept. of the Navy, 1972.

U.S. Navy Diving Manual. NAVSEA 0994-LP-001-9010. Volumes 1 and 2. Includes changes 1 and 2. Washington: Navy Department, Jan 1979.

U.S. Navy Diving Manual. NAVSEA 0994-LP-001-9020. Volume 2, Revision 1. Washington: Navy Department, Jul 1981.

Weihe, W.H. The physiological effects of high altitude. New York: Macmillan, 1964.

Weybrew, B.B. and E.M. Noddin. Psychological effects of prolonged exposure to sonar signals at an elevated intensity. II. Twenty-four days' exposure to signals at 85 dB. NSMRL Rept. 691. Groton, CT: Naval Submarine Med. Res. Lab., Dec 1971.

Workman, R.D., G.F. Bond and W.F. Mazzone. Prolonged exposure of animals to pressurized normal and synthetic atmospheres. NSMRL Rept. 374. Groton, CT: Naval Submarine Med. Res. Lab., Jan 1962.

Wright, W.B. Use of the University of Pennsylvania, Institute for Environmental Medicine procedure for calculation of cumulative pulmonary oxygen toxicity. Report 2-72. Washington: U.S. Navy Experimental Diving Unit, 1972.

Yount, D.E. Multiple inert gas bubble disease: A review of the theory. In: Isobaric inert gas counterdiffusion, edited M.B. Kent. Preliminary report to Office of Naval Research under contract N00014-C-0319. Bethesda, MD: Undersea Medical Society, 1982.

APPENDIX A.
CONDENSED LOGS OF SHAD AND NISAT DIVES

These logs have been extracted from the original working logs, except the Pre-SHAD log which was taken from a handwritten extract of the original. The original working logs followed the format of the International Decompression Data Bank (University of Pennsylvania; Bardin, 1973) but were changed here to make them more readable.

We consider "day 1" as the 24-hour calendar day (midnight to midnight) on which the dive was started, consequently "dive days" coincide with calendar days regardless of what time of day the dive may have been started.

The log of SHAD I was originally recorded against "dive time," which started at 1139 hours on 1973 October 1. This time was followed on a dive time clock that was started at that moment and maintained throughout the dive. This made it easy enough to record each entry at the time against that clock, but created immense problems thereafter because each SHAD I time entry was offset from real time by 11 hours and 39 minutes. Further, early morning events during the second day of the dive were still considered part of "Dive Day 1," and so on. After this dive we realized that a "time of day" log is far more practical and on the occasions when "dive time" is needed it can be calculated by applying the initial offset to the time of the event in question. To convert the dive time log of SHAD I to time of day we constructed a simple minute-by-minute nomogram covering 24 hours for quick conversion of the SHAD I logs times to actual time of day.

All logs follow the same format. The "environmental" information in each entry includes the time of day, depth in feet of sea water, the oxygen partial pressure in atmospheres, the temperature in degrees Fahrenheit, and the percent relative humidity, all as measured in the inner lock. Oxygen levels were recorded in the original logs as oxygen percentages, but have been converted to partial pressures here. Carbon dioxide level was not included as a regular entry in the logs but was recorded regularly. We have put CO_2 entries in the remarks; this parameter never exceeded innocuous levels.

The "environment" for these dives did not change very much. For this reason and to save space we have included only occasional entries that show just the environment, and have tried to include this information about every quarter day. Blank or missing entries indicate no information was available. Some environmental parameters are also shown graphically in section II.G and the frontispiece. An oxygen exposure log is given in Chapter VII.

Comments in the logs describe events such as excursions, special events such as the gas switching in Nisat/He II and III, Doppler bubble counts, unusual occurrences, and the locking into the chamber of

investigators, physicians and visitors. Times of visitor "dives" are from leaving the surface until the return to surface pressure, and may encompass decompression stops.

We have also included symptoms of any sort reported by the divers, results of any medical examinations which may have been logged and any medications logged (except the drugs used for eye dilation for fundus photography). The term "vital signs" (or "VS") is entered to show where these were taken; the results are analyzed elsewhere so we have not included them here. All occasions of Doppler readings after decompression and gas switching are included, whether or not bubbles were heard; (more about Doppler in VI.B). Symptoms, Doppler signals, etc., which were loosely quantified on a "scale of one to ten" are shown as fractions, e.g. 3/10.

We have not included most routine housekeeping such as meals, lock transfers and CO_2 absorbent cannister changes, nor the normal performance of the many scheduled tests and measurements. Logged exercise periods are all included, but the time covered includes pre- and post-run monitoring as well; the actual exercise periods were 20 minutes. Comments are recorded literally in many cases, but are condensed where space could be saved without changing the meaning. Times for a few of the events are included in the comments.

The excursions are described by a remark inserted at the time of day the excursion started. These include the depth of the excursion in fsw, the travel time going to the excursion depth (tvl), the time on bottom (bt), and the total time of the excursion in minutes from leaving storage depth to return to storage depth (tt). This gives all information available about the profile of the excursion in a compact form (ascent time is available by subtraction). This made it easier to do computer analysis of the oxygen exposure.

day	fsw	atm	R.H.%	Remarks
* * * * *	1973 September 10, Dive Day 1 * * * * *			
1235	0	0.21		Begin descent; divers GA and TT.
1236	50	0.53	80/78	Reach 50 feet.
1452	50	0.51	78/78	Divers report chamber is stuffy.
1608	50		78/78	Divers request water, report chamber is hot.
1635	50		82/96	Pulmonary function tests complete.
1835	50		78/82	Dinner at 1936.
2128	50	0.51	80/74	GA requests hygrometer to test humidity; feels humidity is inaccurate.
2400	50	0.51	78/78	PCO_2=1.1 mmHg.
* * * * *	1973 September 11, Dive Day 2 * * * * *			
0308	50	0.52	76/82	PCO_2=1.1 mmHg.
0905	50	0.52	77/74	Both divers report cool sensation, still clammy.
0954	50	0.52	78/79	Divers feeling comfortable. Pulm. fn. tests.
1600	50	0.51	88/71	Pulmonary function tests.
1900	50	0.50	79/74	Dinner. PCO_2=2.1 mmHg.
2400	50	0.50	78/78	
* * * * *	1973 September 12, Dive Day 3 * * * * *			
0300	50	0.50	78/78	PCO_2=0.6 mmHg.
0848	50	0.51	76/74	Begin decompression; travel rate 10 fsw/hr (6 min/fsw); Pulmonary function tests.
1148	20	0.33		Travel rate 4 fsw/hr (15 min/fsw).
1229	17	0.31		No Doppler bubbles.
1340	12	0.28	77/63	GA reports slight headache.
1412	10	0.26		GA reports both divers checked with Doppler monitor; TT has 0, GA about 2/10.
1418	10	0.26	77/66	Travel rate 3 fsw/hr (20 min/fsw).
1436	9	0.26	78/62	GA reports headache clearing; right knee stiff, neurological exam normal.
1553	5	0.23		Doppler TT 1/10 from left arm; GA 3/10, r.leg.
1558	5	0.23	79/62	Travel rate 2 fsw/hr (30 min/fsw).
1700	3	0.23	80/62	Doppler TT 0; GA 3/10.
1822	0	0.21		Reach surface.
1830	0	0.21		GA possible hit; mildly aching knees, throbbing slightly; general ill-at-ease feeling; blood pressure 124/74, pulse 84. TT blood pressure 112/76, pulse 88, slight leg discomfort.
1939	0	0.21		GA reports ache in knees about the same, walking difficult; blood pressure 112/64; temp 98.9F. TT feels a bit achy like "flu" in muscles below the waist; blood pressure 108/74, temp 100.5F; has had DCS, thinks this is not a hit.
2030	0	0.21		GA rectal temp 100.5F, oral 99F.
2128	0	0.21		TT symptoms partly relieved after warm shower; more ache in left knee than right; minimal radiation to left hip; oral temp 100.5F. GA feels better after warm shower, left knee normal, slight aching in right knee; b.p. 112/64, oral temp 99F. GA and TT: neurology normal, strength and range of movement normal for all extremities. No rash or skin changes.
2200	0	0.21	75	On O_2, begin 2 min compression to 60 fsw.
2202	60	0.59		TT relief; mild residual back of left leg.
2204	60	0.59		GA no changes.
2212	60	0.59		TT right knee fine, left knee better; GA both knees fine.
2216	60	0.59		Divers report significant improvement; physician elects treatment Table 5.
2222	60	0.59		Off O_2.
2227	60	0.59		On O_2. TT still tender below right knee. GA left knee painful and right knee tender.
2247	60	0.59		Off O_2. Physician changes treatment to Table 6.
2252	60	0.59		On O_2.
2312	60	0.59		Off O_2. TT no change. GA slight improvement.
2317	60	0.59		On O_2. Depart 60 fsw.
2347	30	0.40		Off O_2. Divers report improvement of knee pain.
2354	30	0.40		TT improved. GA dull pain below left knee.
* * * * *	1973 September 13, Dive Day 4 * * * * *			
0002	30	0.40		On O_2. GA breathing difficult, sleepy.
0038	30	0.40		GA no symptoms. TT pain diminishing in knees.
0102	30	0.40		Off O_2.
0117	30	0.40		On O_2. GA cramp in left calf. TT residual tenderness.
0204	30	0.40		GA mouth tingling (lasts 9 min); off O_2 early.
0217	30	0.40		Depart 30 fsw, to sfc at 30 fsw/min.
0218	0	0.21		Chamber reaches surface, divers exit, TT off O_2.
0243	0	0.21	75	GA reports mild tenderness behind left knee and dull sensation in right calf TT reports right leg normal, left knee has mild discomfort.
0250	0	0.21		GA coughs, especially with deep breath; feels tightness or fullness in chest. TT tightness in chest and coughs with deep breath.
0800	0	0.21		Divers report mild residual tenderness in knees.
1200	0	0.21		GA deep breathing painful, right knee aches.
2300	0	0.21		TT left leg aches at bedtime.
* * * * *	1973 September 14, Postdive Day 1 * * * * *			
0700	0	0.21		GA feels good on arising. TT slept poorly. Both have mild residual pain in knees.
1000	0	0.21		TT left knee and hip painful, walking difficult.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
* * * * * 1973 October 1, Dive Day 1 * * * * *				
1139	0	0.53	76/	Begin pressurization, divers WB and SW.
1142	50	0.53	76/	Reach bottom.
1709	50	0.52	79/54	
2209	50	0.51	73/60	
* * * * * 1973 October 2, Dive Day 2 * * * * *				
0639	50	0.50	72/	
0727	50	0.53	72/62	Lock in MD (RW) and physiologist (AM), 90 min.
1209	50	0.52	76/64	
1339	50	0.51	77/64	
1709	50	0.51	77/62	
2209	50	0.50	78/64	
* * * * * 1973 October 3, Dive Day 3 * * * * *				
0639	50	0.50	76/65	
0741	50	0.50	76/72	Lock in MD (CH) with breakfast, for 55 min.
1115	50	0.51	79/64	Divers begin exercise, 30 min each.
1839	50	0.50	83/67	
2239	50	0.50	81/	
* * * * * 1973 October 4, Dive Day 4 * * * * *				
0439	50	0.50	73/	
0745	50	0.50	75/74	Lock in MD (RW) for 35 min.
1105	50	0.53	79/62	Divers begin 100W exercise, 15 min.
1218	50	0.53	79/62	Lock in dentist (JB) for 36 min.
1741	50	0.53	78/58	Dinner.
2339	50	0.50	77/	
* * * * * 1973 October 5, Dive Day 5 * * * * *				
0409	50	0.51	73/63	
0714	50	0.51	73/	SW and WB complaining of facial rash.
0753	50	0.51	73/71	Lock in MD (CH) and AM for 55 min.
1008	50	0.51	75/66	Lock in BC for 48 min.
1809	50	0.53	76/60	Movie.
2309	50	0.51	76/66	
* * * * * 1973 October 6, Dive Day 6 * * * * *				
0539	50	0.51	72/	
0814	50	0.51	74/	Lock in MD (RW) and physiologist for 81 min.
1153	50	0.51	71/65	Diver WB exercises 28 min.
1711	50	0.51	74/59	
2136	50	0.50	75/69	Lock in communications specialist (NC), 73 min.
2318	50	0.50	76/74	
* * * * * 1973 October 7, Dive Day 7 * * * * *				
0439	50	0.50	75/	
0740	50	0.50	77/63	Lock in MD (WT) for 71 min.
1011	50	0.50	74/62	Exercise 100w, 15 min SW, 11 min WB.
1839	50	0.50	74/64	Dinner.
2309	50	0.50	79/84	
* * * * * 1973 October 8, Dive Day 8 * * * * *				
0539	50	0.50	78/	
0734	50	0.50	79/82	Lock in MD (RW) for 73 min.
1133	50	0.50	78/63	Lock in physiologist (GA) for 63 min.
1832	50	0.50	77/62	
2309	50	0.50	78/60	
* * * * * 1973 October 9, Dive Day 9 * * * * *				
0609	50	0.50	79/	
0757	50	0.50	78/65	Lock in MD (RW) for 42 min.
1015	50	0.50	76/62	Excursion to 200 fsw; tvl=3, bt=18, tt=26 min.
1038	50	0.50	80/	Doppler SW 4 min, WB 3 min, no bubbles heard.
1050	50	0.50		Doppler SW 3 min, WB 2 min; SW 3 min @ 1105.
1159	50	0.50	75/	Doppler WB 2 min, SW 2 min, nothing reported.
1619	50	0.51	77/57	Last Doppler 2 min each, no bubbles reported.
2318	50	0.51	76/74	
* * * * * 1973 October 10, Dive Day 10 * * * * *				
0609	50	0.50	75/	
0751	50	0.50	77/68	Lock in MD (RW) and physiologist (AM), 82 min.
1042	50	0.51	76/62	Exercise, SW 15 min, WB 20 min.
1814	50	0.50	78/60	
2239	50	0.50	76/60	
* * * * * 1973 October 11, Dive Day 11 * * * * *				
0609	50	0.52	75/	
0750	50	0.51	76/61	Lock in MD (RW) for 63 min.
1029	50	0.52	75/67	Exercise, SW 15 min, WB 20 min.
1338	50	0.51	75/67	Lock in GS for 82 min.
2339	50	0.51	76/60	
* * * * * 1973 October 12, Dive Day 12 * * * * *				
0639	50	0.50	78/61	
0749	50	0.51	76/63	Lock in MD (CH) for 54 min.
1013	50	0.51	76/	Excursion to 235 fsw; tvl=3, bt=9, tt=15 min. Doppler on return, SW 1 min, WB 2, SW 2 min.
1036	50	0.52	74/	Doppler SW 1 min, WB 2 min; SW 1 min at 1102, no bubbles reported.
2339	50	0.50	77/59	

Day	Depth fsw	atm	R.H.%	Remarks
* * * * * 1973 October 13, Dive Day 13 * * * * *				
0409	50	0.50	77/	
0731	50	0.50	79/56	Lock in MD (WT) for 39 min.
1042	50	0.51	75/59	Exercise, SW for 19 min, WB for 20 min.
1852	50	0.51	76/59	
2339	50	0.50	76/	
* * * * * 1973 October 14, Dive Day 14 * * * * *				
0609	50	0.50	78/56	
0748	50	0.50	77/	Lock in MD (RW) and physiologist (AM), 82 min.
1009	50	0.50	76/	Excursion to 235 fsw; tvl=3, bt=6, tt=16 min. Doppler SW 1 min, JB 2 min; SW 2 min @ 1022, 10 min @ 1028, 2 min @ 1134; WB 5 min @ 1028, 2 min @ 1036, 1 min 1134; no bubbles reported.
1208	50	0.50	78/	Lock in visitors SS & PG for 12 min. PCO2=1.1.
2201	50	0.50	76/60	Test BIBS on emergency air, system OK.
* * * * * 1973 October 15, Dive Day 15 * * * * *				
0639	50	0.51	79/65	PCO2=1.0 mmHg.
0831	50	0.51	78/63	Lock in MD (RW) for 63 min.
1152	50	0.53	75/61	Exercise, WB for 29 min. PCO2=0.4 mmHg.
1740	50	0.50	76/60	PCO2=0.5 mmHg.
* * * * * 1973 October 16, Dive Day 16 * * * * *				
2409	50	0.52	79/	Taps. PCO2=1.0 mmHg.
0640	50	0.51	79/55	PCO2=0.5 mmHg.
0923	50	0.51	78/65	Lock in MD (RW) for 35 min.
1140	50	0.52	75/	SW fell on left elbow and left knee while repairing lights. PCO2=1.0 mmHg.
1505	50	0.51	77/	Excursion to 235 fsw; tvl=3, bt=7, tt=15 min. Doppler on ascent WB 2 min, SW 1 min, WB 2 min, SW 2 min; SW 3 min @ 1523, 2 @ 1530, 3 @ 1546; WB 3 @ 1550; no bubbles reported.
2347	50	0.51	77/61	Lights out. PCO2=0.8 mmHg.
* * * * * 1973 October 17, Dive Day 17 * * * * *				
0640	50	0.51	80/54	PCO2=0.8 mmHg.
0750	50	0.52	78/60	Lock in MD (CH) for 63 min.
1011	50	0.51	76/61	Excursion to 200 fsw; tvl=3, bt=18, tt=26 min. Doppler SW 2 min @ 1030, 1034, 1038, 1054; WB 1 min @ 1032, 1053; 2 min @ 1036, 1040; no bubbles reported.
1225	50	0.52	79/61	Lock in GS for 55 min. PCO2=0.7 mmHg.
1323	50	0.52	77/	Lock in MD (RW) and MS for 67 min.
2339	50	0.51	77/	PCO2=0.8 mmHg.
* * * * * 1973 October 18, Dive Day 18 * * * * *				
0634	50	0.52	77/56	PCO2=0.7 mmHg.
0752	50	0.51	78/63	Lock in MD (RW) and physiologist (AM), 56 min.
1209	50	0.52	77/	PCO2=1.0 mmHg.
1739	50	0.51	75/60	PCO2=0.4 mmHg.
2339	50	0.50	77/59	PCO2=0.8 mmHg.
* * * * * 1973 October 19, Dive Day 19 * * * * *				
0639	50	0.50	77/59	PCO2=0.4 mmHg.
1209	50	0.51	78/60	PCO2=0.7 mmHg.
1505	50	0.50	76/	Excursion to 200 fsw; tvl=3, bt=19, tt=27 min. Doppler SW 1 min @ 1527, 5 @ 1530, 4 @ 1611; WB 2 min @ 1528, 1535, and 1551; no bubbles heard.
2009	50	0.51	80/63	PCO2=0.4 mmHg.
* * * * * 1973 October 20, Dive Day 20 * * * * *				
0210	50	0.50	76/	PCO2=0.1 mmHg.
0800	50	0.50	77/68	Lock in MD (RW) and physiologist (AM), 79 min.
1342	50	0.50	76/60	Lock in BC for 81 min. PCO2=0.6 mmHg.
1700	50	0.51	77/60	
2310	50	0.51	76/61	Excursion to 200 fsw; tvl=3, bt=16, tt=26 min. Doppler SW 2 min @ 2334, 2338, 0037; 4 min @ 2343, 1 min @ 0008; WB 2 min @ 2336, 2341, 0039; 3 min @ 2350, 1014; no bubbles reported.
* * * * * 1973 October 21, Dive Day 21 * * * * *				
0509	50	0.51	77/	PCO2=0.2 mmHg.
0813	50	0.50	77/61	Lock in MD (WT) for 66 min.
1022	50	0.50	78/61	Excursion to 150 fsw; tvl=2, bt=43, tt=49 min. Doppler SW 2 min 1115, WB 2 min 1117; nothing.
1739	50	0.51	77/68	PCO2=0.6 mmHg.
2339	50	0.50	77/	PCO2=0.7 mmHg.
* * * * * 1973 October 22, Dive Day 22 * * * * *				
0639	50	0.50	79/58	PCO2=0.7 mmHg.
0759	50	0.50	79/	Lock in MD (CH) and physiologist (AM), 77 min.
1014	50	0.50	78/	Excursion to 5 fsw; tvl=3, bt=31, tt=35 min. Doppler SW 3 min 1014, 7 min @ 1027, 5 min @ 1040; 4 min @ 1053 gave 2/10 bubbles provoked by moving arms & legs. WB 9 min @ 1017; 5 min @ 1035 and 8 min @ 1045 gave 3/10 spontaneous bubbles which stopped on ascent at 30 fsw but 2/10 were provoked by moving extremities.
1112	50	0.52	78/	Doppler SW 4 min, WB 2 min, all clear.
2339	50	0.50	77/61	PCO2=0.6 mmHg.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
* * * * * 1973 October 23, Dive Day 23 * * * * *				
0639	50	0.50	77/61	PCO2=0.4 mmHg.
0755	50	0.50	77/61	Lock in MD (RW) for 66 min.
1509	50	0.50	77/63	Excursion to 150 fsw; tvl=2, bt=43, tt=48 min. Doppler SW 1 min ● 1550, 1624, 1632; 4 min ● 1559; WB 8 min 1551, 2 min 1604, 1626; clear.
2349	50	0.51	77/63	PCO2=0.6 mmHg.
* * * * * 1973 October 24, Dive Day 24 * * * * *				
0409	50	0.50	76/	PCO2=0.3 mmHg.
0757	50	0.50	78/77	Lock in MD (WT) and physiologist (AM), 82 min.
0928	50	0.51	79/	Lock in MD's (RS, WB, and CH) for 32 min.
1156	50	0.50	78/63	Lock in GS for 9 min.
2339	50	0.50	76/63	PCO2=0.2 mmHg.
* * * * * 1973 October 25, Dive Day 25 * * * * *				
0709	50	0.50	78/58	PCO2=0.4 mmHg.
0759	50	0.50	77/59	Lock in MD (RW) for 59 min.
1510	50	0.50	78/	Excursion to 15 fsw; tvl=2, bt=57, tt=60 min. Doppler WB 4 min 1509, 1534, 1604, 1 min 1521, 5 min 1553, clear; SW 8 min 1513; 4 min ● 1528 gave 2/10 for 15 sec; 2 min 1538, 3 min 1550, 6 min 1558, 5 min 1608, 3 min 1613, clear.
2307	50	0.51	77/65	Excursion to 150 fsw; tvl=2, bt=49, tt=55 min. Doppler SW 4 min 2356, 0003, 0016; 2 min 0038; WB 3 min 2400, 7 min 0007, 9 min 0028; clear.
* * * * * 1973 October 26, Dive Day 26 * * * * *				
0206	50	0.50	76/	Medical check, both divers OK. PCO2=0.2 mmHg.
0802	50	0.50	77/61	Lock in MD (WT) and physiologist (AM), 20 min.
1016	50	0.51	79/64	Excursion to 150 fsw; tvl=2, bt=43, tt=49 min. Doppler SW 3 min ● 1101, 1106, 5 min 1111, 2 min 1129; WB 1 min 1104, 1133; 3 min 1108, ok.
1213	50	0.51	79/60	Lock in MD (RW) for 13 min.
1457	50	0.50	79/60	Excursion to 75 fsw; tvl=3, bt=60, tt=83 min. Doppler SW 8 min 1607, 2 min 1618, 1633; WB 1 min 1615, 2 min 1631; no bubbles reported.
1632	50	0.50	80/58	Lock in JJ for 52 min.
2339	50	0.51	81/	PCO2=0.6 mmHg.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
* * * * * 1973 October 27, Dive Day 27 * * * * *				
0634	50	0.50	79/60	
0755	50	0.51	78/60	Lock in MD (CH) for 58 min. PCO2=0.4 mmHg.
1014	50	0.51	78/63	Excursion to 100 fsw; tvl=1, bt=58, tt=61 min. Doppler SW 3 min 1117, 1 min 1150; WB 4 min 1120, 1 min 1147; no bubbles reported.
2314	50	0.50	78/60	Excursion to 15 fsw; tvl=2, bt=49, tt=55 min. Doppler WB 3 min 2319, 2 min 2336, 2354, 5 min 0008; WB 5 min 2328, 3 min 2351, 2 min 2405.
* * * * * 1973 October 28, Dive Day 28 * * * * *				
0143	50	0.51	78/62	Medical check, divers OK. Also ok at 0303.
0904	50	0.51	77/	Lock in MD (WT) and physiologist (AM), 81 min.
1113	50	0.51	80/	Excursion to 5 fsw; tvl=3, bt=30, tt=34 min. Doppler WB 5 min 1113, 4 min 1145, 1 min 1237; SW 3 min 1124, 2 min 1140; no bubbles.
1225	50	0.52	81/	Lock in dentist (MF) for 26 min. PCO2=0.7 mmHg.
1605	50	0.51	80/	Excursion to 200 fsw; tvl=4, bt=18, tt=26 min. Doppler SW 3 min 1656, 2 min ● 1735 gave 2/10; 2 min 1853, 4 min 1926; WB 2 min 1700, 1737, 3 min 1855, 2 min 1931; no other bubbles.
1632	50	0.51	83/61	Lock in dentist (JB) for 42 min (began compression at 1628). PCO2=0.3 mmHg.
2339	50	0.50	77/65	PCO2= 0.5 mmHg.
* * * * * 1973 October 29, Dive Day 29 * * * * *				
0730	50	0.50	79/62	PCO2=0.3 mmHg.
0900	50	0.52	79/63	Lock in MD (RW) and dentist (JB) for 80 min.
1523	50	0.50	79/68	Lock in GS for 72 min. PCO2=0.1 mmHg.
1842	50	0.50	77/65	PCO2=0.6 mmHg.
2120	50	0.50	77/	Exercise, WB for 29 min, SW for 29 min.
2339	50	0.50	77/	PCO2=0.6 mmHg.
* * * * * 1973 October 30, Dive Day 30 * * * * *				
0758	50	0.50	79/	Lock in MD (RW) and physiologist (AM), 53 min.
0905	50	0.50		Begin decompression; Travel rate 6 min/fsw.
1035	35	0.43		Travel rate 15 min/fsw (No further environmental entries.)
1650	10	0.27		Travel rate 33 min/fsw
1935	5	0.24		Travel rate 36 min/fsw
2235	0	0.21		Reach surface. No Doppler studies logged; no symptoms reported.

day	fsw	atm	R.H.%	Remarks
* * * * * 1973 March 15, Dive day 1 * * * * *				
1400	0	0.59	71/52	Begin pressurization, divers GS and RF.
1402	60	0.59		Reach bottom. PCO_2=0.
1915	60	0.58	80/40	Exercise 150w, 10 min both divers.
2301	60	0.58	79/47	PCO_2=1.6 mmHg.
* * * * * 1974 March 16, Dive day 2 * * * * *				
0700	60	0.58	82/39	PCO_2=0.9 mmHg.
0722	60	0.57		Lock in MD (RW) and physiologist (AM), 89 min.
1206	60	0.57	82/40	PCO_2=1.1 mmHg.
1419	60	0.57	80/57	Exercise, GS 11 min, RF 9 min.
2141	60	0.56	73/53	Divers submit hair clipping samples.
* * * * * 1974 March 17, Dive day 3 * * * * *				
0700	60	0.56	79/47	PCO_2=1.1 mmHg. Barometer 746 mmHg.
0725	60	0.56		Lock in MD (CH) and SW for 74 min.
1146	60	0.57	75/54	PCO_2=1.1 mmHg. Both exercise 10 min.
1805	60	0.57	73/57	PCO_2=0.8 mmHg.
2300	60	0.56	74/57	PCO_2=0.4 mmHg.
* * * * * 1974 March 18, Dive day 4 * * * * *				
0726	60	0.57		Lock in MD (WT) and physiologist (AM), 84 min.
1212	60	0.57		Lock in SJW for 39 min.
1300	60	0.58	72/53	Exercise 100w, RG 10 min; 125w, GS 10 min.
1828	60	0.58	74/49	PCO_2=1.4 mmHg.
2206	60	0.56	73/53	
* * * * * 1974 March 19, Dive day 5 * * * * *				
0300	60	0.56	72/55	PCO_2=0.9 mmHg.
0748	60	0.57	76/50	Lock in MD (RW) for 69 min.
1207	60	0.57	75/50	Exercise 100w, RF 10 min; 125w, GS 10 min.
1422	60	0.57	74/50	Lock in Chloroseptic throat spray, Cortisporin.
1800	60	0.56	76/48	PCO_2=1.0 mmHg.
* * * * * 1974 March 20, Dive day 6 * * * * *				
0616	60	0.56	73/56	Divers report one cockroach aboard.
0717	60	0.56		Lock in MD (RW) and AM for 73 min.
0740	60	0.58	75/53	RF complaining of sore throat.
0845	60	0.56	73/55	Lock in SW and SJW for 21 min.
0929	60	0.59		Hygrometer reads 71%.
1354	60	0.57	74/51	Exercise, RF 29 min, GS 28 min.
1600	60	0.57	72/57	PCO_2=1.0 mmHg.
1909	60	0.57	72/54	Lock in PG and SS for field day (cleanup).
2300	60	0.57	73/	PCO_2=0.4 mmHg.
* * * * * 1974 March 21, Dive day 7 * * * * *				
0500	60	0.56	73/52	PCO_2=0.2 mmHg.
0719	60	0.56		Lock in MD (WT) and SW for 80 min.
1200	60	0.56	75/49	PCO_2=1.1 mmHg.
1744	60	0.56	75/53	Life support system off for 37 min.
1800	60	0.56	75/58	PCO_2=2.6 mmHg. Vent started at 2.74 mmHg.
2300	60	0.58	72/58	PCO_2=0.6 mmHg.
* * * * * 1974 March 22, Dive day 8 * * * * *				
0200	60	0.58	71/57	GS taking eardrops.
0701	60			Lock in MD (WT) and physiologist (AM), 80 min.
1012	60	0.58	73/55	Excursion to 100 fsw; tvl=1, bt=60, tt=63 min.
1119	60	0.58		Doppler for 9 min, no bubbles reported.
1800	60	0.58	72/53	PCO_2=1.0 mmHg.
2200	60	0.58	74/50	Exercise, 10 min each.
* * * * * 1974 March 23, Dive day 9 * * * * *				
0757	60	0.57	75/52	Lock in MD (CH) and FR for 83 min.
1200	60	0.57	75/49	PCO_2=0.7 mmHg.
1803	60	0.56	77/48	PCO_2=0.4 mmHg.
2200	60	0.56	74/49	PCO_2=1.0 mmHg. Sudafed locked in for GS.
* * * * * 1974 March 24, Dive day 10 * * * * *				
0700	60	0.56	73/48	PCO_2=0.9 mmHg.
0820	60	0.56		Lock in MD (WT) and physiologist (AM), 72 min.
1233	60	0.57	73/50	GS reports itchy scrotum.
1803	60	0.56	74/51	PCO_2=0.8 mmHg.
2312	60	0.57	76/53	PCO_2=0.6 mmHg.
* * * * * 1974 March 25, Dive day 11 * * * * *				
0604	60	0.57	71/55	PCO_2=0.2 mmHg.
0720	60	0.57		Lock in MD (CH) for 83 min.
1140	60	0.57	74/52	Exercise, GS 12 min, RF 10 min.
1216	60	0.58		Lock in LB and JW for 37 min.
1640	60	0.56	74/50	Lock in MD (CH) for 43 min.
1800	60	0.58	74/52	PCO_2=1.0 mmHg.
2303	60	0.57	73/54	PCO_2=1.3 mmHg.
* * * * * 1974 March 26, Dive day 12 * * * * *				
0602	60	0.56	72/50	PCO_2=0.1 mmHg.
0706	60	0.57		Lock in MD (RW) and physiologist (AM), 81 min.
1001	60	0.57	74/50	Excursion to 100 fsw; tvl=1, bt=60, tt=62 min.
1107	60	0.57		Doppler 6 min GS, 3 min RF, no bubbles reported.
1700	60	0.56	76/47	Divers report poor appetites.
2300	60	0.56	74/52	PCO_2=0.4 mmHg.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
* * * * * 1974 March 27, Dive day 13 * * * * *				
0706	60	0.56	73/71	Lock in MD (RW) and SW, 81 min; PCO_2=0.4.
0934	60	0.57	74/52	Exercise, 28 min, both divers.
1306	60	0.57	75/50	Lock in MD's (VB, RS and CH) for 18 min.
1902	60	0.56	72/53	PCO_2=1.0 mmHg.
* * * * * 1974 March 28, Dive day 14 * * * * *				
0200	60	0.56	72/52	PCO_2=0.4 mmHg.
0732	60	0.56	74/56	Lock in MD (RW) and physiologist (AM), 83 min.
0932	60	0.58		Exercise, 10 min each.
1500	60	0.58	76/45	Excursion to 100 fsw; tvl=1, bt=60, tt=62 min.
1604	60	0.58		Doppler for 4 min each, no bubbles reported.
2100	60	0.56	71/52	PCO_2=2.4 mmHg; fooling with scrubbers.
2300	60	0.56	70/55	PCO_2=0.5 mmHg.
* * * * * 1974 March 29, Dive day 15 * * * * *				
0300	60	0.56	70/54	PCO_2=0.2 mmHg.
0814	60	0.56	74/60	Lock in MD (CH) for 57 min.
1016	60	0.56		Exercise 150w, GS 10 min; 100w, RF 10 min.
1200	60	0.58	71/53	PCO_2=1.0 mmHg.
1403	60	0.58		Lock in MD (EF) and physiologist (DM), 28 min.
1800	60	0.56	71/53	PCO_2=0.7 mmHg.
2300	60	0.56	72/52	PCO_2=0.4 mmHg.
* * * * * 1974 March 30, Dive day 16 * * * * *				
0700	60	0.56	73/67	PCO_2=0.7 mmHg.
0703	60	0.56		Lock in MD (RW) and physiologist (AM), 80 min.
1000	60	0.56		Excursion to 150 fsw; tvl=1, bt=60, tt=64 min.
1107	60	0.57	75/54	Doppler, 2 min GS, 3 min RF, no bubbles reported.
1525	60	0.57	74/51	Exercise, 10 min, both divers.
1806	60	0.56	73/55	PCO_2=1.1 mmHg.
2305	60	0.56	73/50	PCO_2=0.8 mmHg.
* * * * * 1974 March 31, Dive day 17 * * * * *				
0742	60	0.56	74/54	Lock in MD (RW) for 68 min.
1156	60	0.57	73/53	Exercise 125w, GS 10 min; 100w, RF 10 min.
1507	60	0.57		Lock in MD (CH) for 22 min.
1715	60	0.58		Excursion to 200 fsw; tvl=3, bt=20, tt=23 min.
1743	60	0.58	77/50	Doppler 7 min each, no bubbles reported.
1803	60	0.57	70/50	Doppler 4 min GS, 2 min RF, no bubbles reported.
2300	60	0.58	72/52	PCO_2=1.4 mmHg.
* * * * * 1974 April 1, Dive day 18 * * * * *				
0716	60	0.57		Lock in MD (RW) and physiologist AM, 112 min.
0934	60	0.57	74/54	Lock in PG for 31 min. Glucose tolerance test on both subjects, 7 blood samples.
1051	60	0.57	74/53	Lock in DL for 99 min.
1257	60	0.57	74/52	Lock in PG for 44 min.
1513	60	0.57	72/53	Exercise, GS 27 min, RF 28 min.
2301	60	0.56	69/53	PCO_2=1.0 mmHg.
* * * * * 1974 April 2, Dive day 19 * * * * *				
0709	60	0.59	72/62	Lock in MD (WT) for 97 min.
1015	60	0.56		Excursion to 250 fsw; tvl=5, bt=6, tt=15 min.
1027	60	0.56	71/46	Doppler 8 min GS, 2 min RF, no bubbles reported.
1649	60	0.57	71/48	Exercise, 10 min both divers.
2300	60	0.57	70/51	PCO_2=0.9 mmHg.
* * * * * 1974 April 3, Dive day 20 * * * * *				
0708	60	0.57	70/67	Lock in MD (RW) and physiologist (AM), 73 min.
1000	5	0.23		Excursion to 5 fsw; tvl=4, bt=21, tt=26 min. Doppler 2 min RF, no bubbles reported.
1011	5	0.23		GS slight itching entire body.
1016	5	0.23		RF reports itching of scrotum and forearms.
1018	5	0.23		GS reports red rash and itching on forearms; Doppler for 5 min, grade 3 to 4.
1020	5	0.23		RF reports increased itching on forearms.
1022	5	0.23		RF reports rash on arms and stomach; Doppler for 2 min.
1024	5	0.23		Begin descent; RF dull pain in left ankle.
1025	60	0.57		GS reports itch is diminishing.
1026	60	0.57		Reach 60 fsw; RF on Doppler 2 min, reports itching has stopped. GS itching behind knees only.
1028	60	0.57		Doppler GS 3 min.
1030	60	0.57		GS reports itching and rash on knees only; RF no itching or rash.
1050	60	0.57	74/47	GS reports itching has stopped.
1112	60	0.57	74/47	Doppler 14 min GS, grade 1; grade 2 when stimulated. RF 4 min, no bubbles reported.
1200	60	0.57	72/53	PCO_2=1.0 mmHg.
1210	60	0.56	72/53	Doppler 2 min GS, no bubbles reported.
1715	60	0.57	71/57	Exercise 10 min, both divers.
2300	60	0.56	69/51	PCO_2=1.1 mmHg
* * * * * 1974 April 4, Dive day 21 * * * * *				
0734	60	0.57	71/54	Lock in MD (WT) for 59 min.
0939	60	0.57	72/51	Lock in MD (RS), JC and SW for 26 min.
1247	60	0.57	72/49	Lunch. Ergometer locked in.
1800	60	0.56	70/52	PCO_2=1.4 mmHg.
2100	60	0.56	77/44	PCO_2=0.7 mmHg. Wet/dry RH=67%.

0706	60	0.56	71/78	Lock in MD (CH) and physiologist (AM), 76 min.
1010	60	0.56	73/57	Excursion to 15 fsw; tvl=2, bt=31, tt=34 min.
1013	15	0.29		Doppler 1 min each diver.
1023	15	0.29		Doppler GS 1 min, no bubbles reported.
1028	15	0.29	72/	GS itch behind right knee. Doppler RF, clean.
1037	15	0.29		GS reports slight itching increase on back of knee and forehead, no Doppler bubbles heard.
1044	60	0.56		After 2 min at 60 fsw divers not itching.
1209	60	0.57	71/53	Exercise 125w, GS 20 min; 100w, RF 10 min.
1500	60	0.59	72/55	Excursion to 200 fsw; tvl=2, bt=20, tt=27 min.
1528	60	0.59	71/52	Doppler, GS 1 and 3 min; RF 3 min, no bubbles reported; Doppler finished at 1542.
2305	60	0.57	74/50	PCO_2=0.7 mmHg; psychrometer RH 71.5%.

* * * * * 1974 April 6, Dive day 23 * * * * *

0716	60	0.58	72/56	Lock in MD (RW) for 34 min.
1000	60	0.58	75/49	Excursion to 150 fsw; tvl=2, bt=60, tt=65 min.
1109	60	0.59	72/44	Doppler GS 4 min, RF 2 min, no bubbles reported.
1500	60	0.59	73/47	Excursion to 100 fsw; tvl=1, bt=60, tt=62 min.
1606	60	0.59	75/46	Doppler, GS 2 min, RF 2 min, GS 2 min, nothing.
2115	60	0.59	72/49	Bilges being pumped; fundus photos cancelled.

* * * * * 1974 April 7, Dive day 24 * * * * *

0704	60	0.59	74/68	Lock in MD (RW) and physiologist (AM), 92 min.
1006	60	0.56		Excursion to 100 fsw; tvl=1, bt=60, tt=62 min.
1112	60	0.58	75/49	Doppler GS 2 min; no bubbles reported.
1312	60	0.58	75/55	Exercise, RF 12 min, GS 10 min.
1500	60	0.58		Excursion to 15 fsw; tvl=2, bt=31, tt=34 min.
1503	15	0.29	72/48	Doppler 1.5 min both divers, no bubbles reported.
1524	15	0.29	73/48	Doppler GS 3 min; RF 1 min at 1525; GS 1.5 min at 1532; RF min at 1540, no bubbles reported.
2300	60	0.58	70/53	PCO_2=0.6 mmHg

* * * * * 1974 April 8, Dive day 25 * * * * *

0727	60	0.58	73/58	Lock in MD (RW) for 104 min.
1000	60	0.59	/50	Excursion to 5 fsw; tvl=3, bt=22, tt=26 min.
1007	5	0.24		Doppler GS 7 min, 1 min at 1012; RF 1 min at 1015, 1 min at 1022, no bubbles reported.
1008	5	0.24		GS slight itch, both forearms, left upper arm.
1011	5	0.24		RF slight itch right arm; GS back itches.
1013	5	0.24		RF itches inside & outside of both arms & legs. GS itches on stomach, no rash as yet.
1016	5	0.24		RF reports pink rash on back and legs; GS reports itch increasing but still slight.
1019	5	0.24		RF rash intensifying, red on periphery of back.
1020	5	0.24		RF and GS report itch increasing to moderate; GS 5/10, RF 6/10.
1023	5	0.24		RF rash inside legs worse; GS itch worse.
1029	5	0.24		Doppler, GS 2 min, 3 min at 1029; RF 2 min at 1034, no bubbles reported. GS rash all over.
1029	60	0.59		GS and RF report relief at 30 feet, rashes decreasing, GS reports pain in left knee.
1035	60	0.59	74/50	GS reports pain has stopped.
1151	60	0.59	71/47	Exercise 100w, RF 9 min, GS 10 min.
1241	60	0.59	74/46	Lock in SW and LK for 49 min.
1409	60	0.56	73/50	Lock in WB and JJ for 55 min.
2300	60	0.59		Excursion to 200 fsw; tvl=2, bt=20, tt=27 min.
2328	60	0.59	79/	Doppler, GS 2 min, RF 2 min at 2333; GS 3 min at 2352; RF 3 min at 0007, no bubbles reported.

* * * * * 1974 April 9, Dive day 26 * * * * *

0912	60	0.56	73/57	Lock in MD (RW) for 60 min.
1540	60	0.58	72/51	Exercise, RF 20 min, GS 28 min.
2300	60	0.57	70/54	PCO_2=0.2 mmHg.

* * * * * 1974 April 10, Dive day 27 * * * * *

0720	60	0.58		Lock in MD (RW), AM and dentist RE, 83 min.
1300	60	0.58	71/51	PCO_2=0.9 mmHg.
2000	60	0.57	72/51	

* * * * * 1974 April 11, Dive day 28 * * * * *

0653	60	0.59	71/63	Lock in MD (WT) and physiologist AM, 73 min.
0800	60	0.59	73/64	Begin decompression; travel rate 6 ft/hr.
1030	48	0.56	72/54	Travel rate 4 ft/hr. Doppler GS, no bubbles.
1236	39	0.45	72/51	Doppler RF, no bubbles reported.
1412	30	0.39	73/49	Doppler both divers, no bubbles reported.
1609	23	0.35	74/48	Doppler GS 2 min, RF 1 min, no bubbles reported.
1745	18	0.31	72/50	Travel rate 2 ft/hr.
1829	17	0.31	73/50	Doppler GS 5 min, RF 6 min, no bubbles reported.
2100	12	0.28	72/51	Hold at depth for 8 hours. PCO_2=1.8 mmHg.

* * * * * 1974 April 12, Dive day 29 * * * * *

0048	12	0.28	72/51	GS reports vague discomfort in left shoulder, noticed since 30 fsw. Doppler GS, no bubbles.
0154	12	0.28	73/51	GS reports vague discomfort in area of eye socket, present only during motion, less noticeable now than at 30 fsw and not increasing. No objective findings of note.
0509	12	0.28	72/51	GS reports no shoulder pain. Resume decompression; travel rate 2 ft/hr.
0706	8	0.25	72/50	Doppler on both divers, no bubbles reported.
0827	6	0.23		GS shoulder "pain" disappeared after shower.
0920	4	0.23	72/60	Doppler OK on both; again at 1011, some suspicious but undefinable sounds on RF.
1100	1	0.21	73/56	GS "pain" similar to last night.
1200	0	0.21		Reach surface.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
* * * * * 1974 December 3, Dive Day 1 * * * * *				
1019	0	0.21		Divers in chamber doing baseline studies.
1042	0	0.21	78/54	Begin pressurization; 3 divers DM, RO and PP.
1044	50	0.53	78/54	Reach bottom.
1752	50	0.52	76/77	Ergometer exercise, PP 28 min.
2051	50	0.52	76/74	Ergometer exercise, DM and RO 25 min.
* * * * * 1974 December 4, Dive Day 2 * * * * *				
0742	50	0.53	79/74	Lock in MD (CH) for 60 min.
1005	50	0.52		Excursion to 100 fsw; tvl=2, bt=478, tt=505 min.
1641	100	0.84	76/75	Lock in MD (JW) for 12 min to draw blood.
1805	100	0.84	76/76	Begin ascent at 2 fsw/min, 25 min to 50 fsw.
1815	80	0.72	76/76	Doppler RO 6 min, PP 3 min, DM 3 min, no bubbles reported.
1830	50			Arrive 50 fsw.
1838	50	0.53	76/76	Doppler RO, DM (1845), RO 1856, 3 min each, no bubbles reported.
1841	50	0.52	76/69	Lock in dentist (RE) for 23 min.
1856	50	0.52		DM reports intermittent pain located at 3rd intercostal space just to left of sterum, not burning in character and not reproduced by external pressure. Pain has been present since about 0900 today, is getting slightly worse but far from incapacitating. No vital capacity changes. No other symptoms.
2057	50	0.52	76/71	Doppler, DM and RO 5 min, PP 3 min, no bubbles.
* * * * * 1974 December 5, Dive Day 3 * * * * *				
0723	50	0.52	76/87	Lock in MD (RW) for 47 min.
1000	50	0.51	76/77	Excursion to 100 fsw; tvl=2, bt=478, tt=500 min.
1010	100	0.83		Weight lifting, RO 29 min.
1119	100	0.83	76/77	Weight lifting, PP 21 min.
1216	100	0.83	75/76	Ergometer exercise, DM 4 min.
1400	100	0.83	76/77	Weight lifting, DM, 30 lbs, 5 lifts per min for 18 min.
1404	100	0.83		Ergometer exercise, RO for 22 min.
1442	100	0.83	78/76	Ergometer exercise, PP 22 min.
1631	100	0.83	79/80	Lock in MD (JW) and RLF for 40 min.
1703	100	0.83	78/77	Lock in AM for 76 min.
1726	100	0.83		Lock in MD (JW) and RLF for 54 min.
1800	100	0.83	77/73	Begin ascent at 2.5 fsw/min, 20 min to 50 fsw.
1827	50	0.52		Doppler DM and RO 3 min each, no bubbles reported. PP 3/10 left arm.
1932	50	0.52	75/69	Doppler PP 2 min, 4/10 left arm. RO clear.
2012	50	0.52		Doppler DM 4 min, 1/10 left side. AM at surface also had bubbles, 5/10 at 1900, 4/10 at 1950, 3/10 at 2039.
2042	50	0.52		Doppler PP 4 min, 1/10 left arm; DM and RO 2 min each, no bubbles.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
* * * * * 1974 December 6, Dive Day 4 * * * * *				
0730	50	0.53	77/81	Lock in MD (CH) for 96 min.
0956	50	0.53	77/74	Weight lifting, PP 16 min.
1000	50	0.53	77/74	Excursion to 100 fsw; tvl=2, bt=478, tt=495 min.
1039	100	0.84	77/76	Weight lifting, PP 10 min.
1141	100	0.84	75/75	Ergometer exercise, RO 30 min.
1148	100	0.84	75/75	Weight lifting, DM 20 min.
1320	100	0.84		Ergometer exercise, PP 28 min.
1631	100	0.84	75/75	Ergometer exercise, DM 28 min.
1800	100	0.83		Begin ascent at 3 1/3 fsw/min, 15 min to 50 fsw.
1813	50	0.53	76/77	Doppler on ascent, PP 3 min, 3/10; DM and RO, no bubbles reported.
1832	50	0.52	75/72	Doppler, PP 1 min, 3/10; RO no bubbles reported.
1855	50	0.52	75/72	Doppler, PP 5 min, 1/10; DM 4 min, 1/10 left leg.
1957	50	0.52	76/71	Doppler, DM 1 min, 1/10 from lower trunk; PP and RO 2 min, no bubbles reported.
2020	50	0.52	76/71	Doppler, RO 2 min, PP 1 min, DM 2 min, clear.
2118	50	0.52	76/71	Doppler, DM 2 min, no bubbles reported.
* * * * * 1974 December 7, Dive Day 5 * * * * *				
0725	50	0.52	77/86	Lock in MD (JW) and AM for 50 min.
0957	50	0.53	77/78	DM weight lifting 20 min.
1000	50	0.52	76/79	Excursion to 100 fsw; tvl=2, bt=478, tt=490 min.
1130	100	0.83	76/83	Exercise, PP 34 min; RO weight lifting 20 min.
1325	100	0.83	76/83	Ergometer exercise DM 27 min, RO 24 min; PP weight lifting 20 min.
1800	100	0.83		Begin ascent at 5 fsw/min, 10 min to 50 fsw.
1807	65	0.61		Doppler PP 3 min, DM 2 min, no bubbles.
1814	50	0.52		Doppler RO 5 min, 2/10 induced by movement.
1820	50			Doppler PP 10 min, DM 3 min, RO 2 min, clear.
1916	50	0.52		Doppler RO 3 min, no bubbles.
1951	50			Doppler PP 4 min, DM 5 min, RP 0.5 min, clear.
2050	50	0.52	79/73	DM reports he is unusually tired with a mild "softball size" substernal discomfort.
2110	50	0.52	79/73	Doppler DM 5 min, no bubbles.
2139	50	0.52	77/72	Lock in MD (RW) for 14 min to examine DM.
2300	50	0.52	76/74	DM reports feeling better.
* * * * * 1974 December 8, Dive Day 6 * * * * *				
0739	50	0.52	76/82	Lock in MD (RW) and physiologist (AM) for 88 min, with Tylenol.
1000	50	0.52	76/76	Excursion to 100 fsw; tvl=2, bt=478, tt=482 min.
1132	100	0.83		Exercise DM 28 min; PP weight lifting 21 min.
1345	100	0.84	74/73	Ergometer exercise RO 22 min, PP 24 min.
1800	100			Begin ascent at 10 fsw/min, 5 min to 50 fsw. Doppler PP 6 min, DM 2 min, RO 1 min, clear.
1815	50	0.52	74/80	Lock in MD for 40 min.
1825	50	0.52		Doppler RO 2 min, PP 1 min, clear; DM 3 min, 2/10 lower trunk.
1852	50	0.52	77/74	Doppler DM 2 min, 2/10 induced; PP 1 min, no bubbles reported.

1918	50	0.52	77/79	Lock in MC for 44 min.
2015	50	0.52	75/70	Doppler DM 2 min, 1/10 lower trunk; PP and RO 1 min, no bubbles.
2145	50	0.52	79/71	DM reports extreme fatigue and metallic taste in mouth beginning at about 2000 hrs. Doppler 5 min, no bubbles.
* * * * * 1974 December 9, Dive Day 7 * * * * *				
0630	50	0.52	76/78	RO reports small finger laceration, washed and treated with tincture Zephiran.
0745	50	0.52	76/78	Lock in MD (CH) for 72 min.
1000	50	0.52	80/60	Excursion to 100 fsw; tvl=2, bt=478, tt=482 min.
1126	100	0.83		Ergometer exercise, RO 39 min.
1500	100	0.83	76/69	Ergometer exercise, DM 30 min
1556	100	0.83	77/71	Ergometer exercise, PP 22 min.
1629	100			Lock in JW and RLF for blood gases; JW 19 min, RLF 35 min.
1652	100	0.83	77/72	Lock in AM for 31 min.
1703	100	0.83	/73	DM reports he has a headache.
1800	100	0.83		Begin 2 min ascent to 50 fsw.
1804	50	0.52		DM headache increased with decompression, still mild. Doppler RO 2 min, clear; DM 2 min, 2/10.
1814	50	0.52	/80	Doppler PP 1 min, no bubbles. Doppler on AM finds no bubbles at 1811, 1/10 at 1833, 1/10 at 1856, clear at 1935.
1836	50	0.52	76/70	Doppler PP 1 min, RO 2 min, clear; DM 1 min, 2/10, lower trunk.
1901	50	0.52		Doppler RO 3 min, PP 2 min, DM 2 min, clear.
1920	50	0.52	76/70	Lock in WB for 18 min.
* * * * * 1974 December 10, Dive Day 8 * * * * *				
0724	50	0.53	76/81	Lock in MD (RW) and physiologist (AM), 71 min.
0942	50	0.52	76/76	Begin decompression; travel rate 10 min/ft.
1212	35	0.42	76/75	Travel rate 15 min/ft. Doppler RO and PP 1 min, DM 2 min, no bubbles reported.
1403	27	0.38	76/74	Doppler DM 4 min, no bubbles reported.
1510	23	0.35	75/73	Doppler PP 2 min, 1/10 flexing knees, RO 2 min, 1/10 flexing knees.
1542	21	0.34	76/74	Decompression rate now 54 min/ft.
1550	21	0.34	76/74	Doppler DM 2 min, clear.
1618	20	0.33	76/74	Doppler PP 3 min, 2/10 flexing knees; DM 4 min, no bubbles.
1719	19	0.33		Doppler RO 1 min, DM 2 min; PP 2 min, 2/10 while doing deep knee bends.
1814	18	0.32	76/73	PP reports pain in knee, consulting with Dr. CH.
1815	18	0.32		Hold depth, ordered by Dr. CH.
1825	18	0.32		Lock in MD (CH) for 103 min.
1834	18	0.32		MD (CH) reports condition of PP, tenderness in right knee, cannot take weight, tends to give out when standing. DM and RO have no symptoms. PP's BP 104/72, pulse 76 regular.
1836	18	0.32	77/73	Recompress 10 fsw.
1842	28	0.38	77/73	PP's knee feeling slightly better, 3/10.
1902	28	0.38		PP on O2 for 10 min.
1912	28	0.38		PP reports complete relief.
1917	28	0.38		PP on O2 for 10 min.
1929	28	0.38		Symptom check on PP; no signs in chest from O2, minimal residual soreness, no symptoms of bends; pulse 64, basal.
1932	28	0.38		PP on O2 for 10 min, residual soreness in knee at end of O2.
1947	28	0.38		PP on O2 for 10 min.
2004	28	0.38	77/76	PP mild soreness in knee when flexed, no pain when knee is straight out; soreness in medial and lateral "coligaments" of knee. Palpation: posterior borders medial and lateral "coligaments" tender; when knee stretched, nothing noticed. Standard tension and flexion evokes no symptoms.
2017	28	0.38		Resume decompression, 54 min/fsw.
2134	26	0.37	77/82	Doppler, DM 2 min, no bubbles heard.
2330	24	0.36		Hold depth until 0600; medical check, no complaints from crew.
* * * * * 1974 December 11, Dive Day 9 * * * * *				
0200	24	0.36	78/73	Diver status check, all OK, no symptoms.
0600	24	0.36	78/72	Resume decompression, rate 54 min/fsw.
1019	19	0.33	78/70	Doppler, RO and PP clear, DM possible 1/10.
1410	14	0.29	78/74	Doppler, 1 min each, no bubbles.
1723	12	0.28		Lock in MD (CH) for 84 min. PP has upper pharyngeal, uvual, and turbinate congestion. Vocal cords not swollen but increased in mucus. Slight cervical node tenderness under angle of mandible. Chest clear with normal expansion; BP 118/80, pulse 68, rr 16, rectal T 99.0F. RO no problems, BP 118/80, pulse 68, rr 16, temp 99.0F. DM has tender chest area, slight discomfort in deep inspiration below clavicle and under sternum; lateral tenderness radiating toward sternum, accentuated by deep breathing; one right cervical gland tender; normal pharynx, mild hacking cough.
2141	6	0.25		Doppler all 3, no bubbles.
2145	6	0.25	78/72	Hold depth overnight.
2214	6	0.25	78/83	Breaker tripped, power to chamber lights and equipment rack off 30 sec.
* * * * * 1974 December 12, Dive Day 10 * * * * *				
0600	6	0.25		Resume decompression, 54 min/fsw.
0635	6	0.25	82/75	RO reports body weight scale may be slightly out of adjustment (he dropped it).
0800	0			Reach surface.
0822	0	0.21		Doppler RO clear, DM 1 possible bubble, PP 1/10 left leg.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
* * * * * 1975 March 3, Lab Day 1 * * * * *				
				Predive human factors training. Diver physicals NT, JB, RJ.
* * * * * 1975 March 4, Lab Day 2 * * * * *				
				Laboratory work.
* * * * * 1975 March 5, Lab Day 3 * * * * *				
				Pre-dive training, lab work, X-ray on JB.
* * * * * 1975 March 6, Lab Day 4 * * * * *				
				Fasting blood samples, dental, training.
* * * * * 1975 March 7 * * * * *				
				Exercise, 100 watts.
* * * * * 1975 March 8, Lab Day 5 * * * * *				
				Blood samples.
* * * * * 1975 March 9 * * * * *				
				Urine sampling continues.
* * * * * 1975 March 10 * * * * *				
				Pre-dive physicals. Medical approval.
* * * * * 1975 March 11, Lab Day 6 * * * * *				
				Blood samples. JB urine has pus cells.
* * * * * 1975 March 12, Dive Day 1 * * * * *				
0700				Throat and urine culture on RJ, feels OK; urinanalysis on JB.
1000				Compression delayed.
1437	0			Begin descent; 3 divers JB, RJ and NT, on mask.
1439	50			Hold at 50 fsw for 14 min for checks.
1500	108			NT reports intermittent pain in left lower molar; familiar metallic taste in mouth.
1513	198			Reach bottom. Intensive testing next 2 hr.
1534	198			Divers remove masks in turn over 12 min.
1747	198	0.24	77/65	JB nauseated.
1752	198	0.24		Exam of JB reveals normal cortical function (interpretation, memory, language), normal effort & attitude; cyanosis. Had nausea and subjective vertigo when eyes closed.
1815	198			JB vomiting.
1835	198			JB and RJ feel cold and clammy.
1840	198			NT felt nauseated while being tested.
1902	198	0.23	76/54	NT nauseated, vomiting. RJ good appetite despite nausea.
1925	198			All divers report cold and clammy feeling; NT had good appetite but lost it and feels somewhat seasick.
1954	198	0.23	77/82	NT vomiting; still feels clammy.
2018	198	0.22	78/54	NT reports feeling better after lying down.
2024	198			NT vomiting; JB awakens, says he is OK.
2027	198			NT reports feeling nauseated when lying down and eyes closed and feeling queasy when standing; pulse normal. Carbon monoxide 2.6 ppm.
2031	198			NT mouth feels dry, has trouble taking own pulse; RJ mouth feels wet; JB sleeping.
2040	198			RJ reports NT looks OK; pulse 58, pupils small.
2044	198			RJ checking JB, pulse 56; pupil response OK.
2045	198			NT reports feeling nauseated, cold and narcotized, speech slurred, having difficulty remembering recent events.
2049	198	0.22	79/65	NT & JB sleeping; RJ feels OK, drinking lots of coffee.
2103	198			NT and JB awakened and say they are feeling better.
2215	198			JB feels nauseated during human factors test.
2230	198			JB reports he has excess gas. CO_2 = 1.3 mmHg.
2240	198			JB and NT vomiting. RJ is OK.
2305	198	0.22	77/53	NT feels cold and nauseated, had chills.
2312	198			JB vomiting. CO_2 = 1.3 mmHg.
2355	198	0.22		Decision to add O_2, may ascend to 165 fsw.
* * * * * 1975 March 13, Dive Day 2, Lab Day 7 * * * * *				
0039	198			RJ speech less slurred "after addition of O_2." PCO_2 not logged.
0058	198	0.28	78/53	PCO_2=2.1 mmHg
0118	198	0.29		JB and NT report feeling better; some residual nausea; all alert and in good moods. PCO_2=2.4.
0211	198	0.31	78/52	NT vomiting; RJ complaining of abdominal discomfort.
0222	198			MD (DK) states NT feels better, some nausea.
0307	198	0.31	78/52	Divers retire. PCO_2=2.4 mmHg.
0326				Taps. Divers slept soundly.
0810	198	0.31		Reveille, VS, some tests before breakfast at 1013.
0900	198			NT queezy, locquacious, not as much nausea, still clammy; RJ has shoulder pains and popping, get Tetralac for stomach; JB arthritis of shoulder.
1240	198	0.29		RJ feels tired, wants to sleep, feels drained, has cold sweat; air seems "thicker."
1326	198	0.28	76/	JB nearly fell asleep doing VER. PCO_2=1.3 mmHg.
2035	198	0.30	78/63	Divers report inside thermometer reads 87. PCO_2=1.1 mmHg.

2215	198	0.30	78/63	NT reports difficulty breathing; RJ reports sore throat. Tetralac, Sudafed in. PCO_2=1.3.
2327	198	0.31	77/63	Lights out.

* * * * * 1975 March 14, Dive Day 3, Lab Day 8 * * * * *

0632	198	0.30	77/59	Reveille. NT chest OK, head stuffed up. VS. PCO_2=0.5 mmHg.
0713	198	0.30	77/54	Divers feel generally good but slightly drunk.
1143	198	0.30	79/57	JB reports headache and nausea.
1240	198			JB headache gone, nausea almost gone, no appetite.
1800	198	0.31	79/60	Order given to switch to decaffinated coffee, except 1st mug in AM. RJ exercising.
2300	198	0.31	78/60	Taps. Divers OK.

* * * * * 1975 March 15, Dive Day 4 * * * * *

0630	198	0.30	76/58	Reveille. Vital signs 0700. Tests all day.
1406	198	0.30	78/56	JB hands numb, speech slurred, feels drunk. NT speech slurred, blase attitude, trouble holding on to things. RJ depressed.
1800	198	0.30	76/59	PCO_2=0.5 mmHg.
2315	198	0.30		JB feels tired, warm and clammy. VS.
2321	198	0.30	74/60	NT feels cold and clammy. Taps (XX)4.

* * * * * 1975 March 16, Dive Day 5, Lab Day 9 * * * * *

0627	198	0.30	75/59	Reveille. Blood draw. PCO_2=0.5 mmHg. RJ headache and dizzy. JB feels good; NT much better, still clammy, slight nausea. All in good spirits.
0908	198		75/60	RJ takes Tylenol.
1204	198	0.30	79/58	Morning lethary followed a very large breakfast.
1527	198	0.29	77/61	Exercise RB 21 min, RJ 28 min, NT 20 min.

* * * * * 1975 March 17, Dive Day 6 * * * * *

0030	198	0.34	76/67	Taps. PCO_2=1.0 mmHg.
0655	198	0.29	75/64	RJ has trouble catching breath. VS.
1510	198	0.30	78/62	Exercise, RJ 29 min, difficulty breathing at start.
1607	198	0.30	78/62	NT exercise; intermittent due to nose clip, EEG. At 1659 aborted after 30 min exercise; "subject failure;" had "crawling feeling in skin, voices sounded distant" a few seconds before.
1743	198	0.30		JB exercise, felt cold; headache and low energy level, bag hard to breathe on. NT has dyspnea on minor exertion while squeezing Douglas bag.
2212	198	0.30	75/62	JB and RJ request Tetralak for upset stomach from pizza. Lights out at 2231.

* * * * * 1975 March 18, Dive Day 7 * * * * *

0630	198	0.30	76/59	Reveille 0700. All report "air feels heavy."
0806	198	0.30		NT complains of left external ear infection; JB reports lips are numb.
1105	198	0.30	80/59	NT instilled otic drops in left ear.
1501	198	0.30	77/60	Exercise, RJ 29 min; NT 24 min; RJ at 1711, 50w, 28 min.
1603	198	0.31		BJ reports he caught a cold previous night.
1711	198	0.32	78/62	RJ complains of lower back pain starting after riding bike for exercise.
1837	198	0.29		All complains of heavy breathing. RB 4 antacid.
1932	198			NT reports inflammation on arm at cut.
2123	198		78/89	All report heavy breathing. Inside RH=89.
2125	198			Reducing temperature and humidity
2206	198		77/62	All three continue breathing difficulty.
2227	198	0.30	77/60	RJ and BJ take antacid for upset stomach "from pizza."
2318	198	0.30		JB complains of headache.
2338	198		77/	Plastic bag caught in life support duct of inner lock causing problems with heat and CO_2.
2345	198	0.30	76/58	NT reports all are breathing better. Taps 2354.

* * * * * 1975 March 19, Dive Day 8 * * * * *

0700	198	0.30	75/61	Reveille. NT mild difficulty breathing, ear drops, left ear 0752. CO_2=1.1 mmHg.
1229	198	0.30	74/63	NT ear drops; Tetralac for RJ.
1432	198	0.30	75/63	RJ reports constant lower back pain since reveille; red spots on upper arm. JB has marks due to sphygmomanometer after 28 hr.
1437	198	0.30		Begin decompression; travel rate 6 fsw/hr.
1617	186	0.29	78/60	Travel rate 4 fsw/hr.
1700	183	0.29	79/60	NT put drops in left ear.
1854	178	0.30	79/59	Travel rate 33 min/fsw.
1911	177	0.30	77/67	Travel rate 52 min/fsw.
2003	176	0.29	77/62	Travel rate 53 min/fsw.
2135	174	0.29		NT drops in left ear.
2202	174	0.29	77/61	NT complains still breathing hard; RJ back still hurts. Taps 2210.

* * * * * 1975 March 20, Dive Day 9, Lab Day 11 * * * * *

0530	165	0.29		RJ says he broke skin on forehead yesterday when he fell. [Fall not entered in log.]
0629	164	0.29		RJ feeling cold and clammy.
0711	163			NT reports some heavy breathing, not as marked; all three divers have cold and clammy feeling; JB complains of pain in lower spine.
0818	162			NT scraped his foot while taking shower; cleaned with bacitracin ointment.
1102		0.35	77/63	Tests proceeding all day. BJ interview with BW.
1544	153	0.30	76/59	Doppler JB 22 min, NT 3 min, no bubbles.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
1714	152	0.31	76/58	Doppler NT 17 min, JB 4 min, no bubbles.
1800	151	0.30	78/58	
2000	129	0.31	76/58	Verbal argument between RJ and JB; light physical contact lasting 5 min.
2201	147	0.39	78/58	
* * * * *	1975 March 21, Dive Day 10 * * * * *			
0330				Divers awakened to walk around, no complaints.
0731	136	0.42	76/57	Reveille.
1200	129		77/61	Real coffee.
1542	126	0.25	78/61	Doppler RJ 8 min, JB 6 min, NT 7 min, clean.
1614	126	0.24	76/57	RJ says he has an infection on his foot.
* * * * *	1975 March 22, Dive Day 11, Lab Day 12 * * * * *			
0008				NT eardrops. Taps 0024.
0700	109	0.31	76/57	Divers awake, feel fine.
1252	102	0.31		MD (CH) reports transient episode of itching on body and arms of NT and JB lasting 10 min.
1331	102	0.30	80/59	Doppler all divers, no bubbles reported.
1850	96	0.30	76/59	Divers complain of stuffiness and difficulty breathing. Testing continues.
1953	95	0.31	75/60	Divers feel hot.
2223	92	0.30		Divers feel cold and clammy; breathing heavily.
2232	91	0.31	75/53	Doppler NT 1 min, JB 2 min, RJ 2 min, clear.
2337	87	0.30	75/59	Taps.
* * * * *	1975 March 23, Dive Day 12 * * * * *			
0917	79	0.30	71/51	Reveille. VS.
1221	75	0.29	75/74	Testing continues.
1646	72	0.30	76/62	Doppler JB 3 min, no bubbles. VS 1900.
2200	65	0.30		
* * * * *	1975 March 24, Dive Day 13 * * * * *			
0403	61	0.30	75/55	Taps.
0630	56	0.30	75/52	Reveille. VS.
1231	41	0.28		RJ ear drops. Testing.
2009	40	0.29	76/50	Doppler RJ 1 min, JB 1 min, NT 4 min, clear.
* * * * *	1975 March 25, Dive Day 14 * * * * *			
0750	27	0.30	75/45	First test 0707.
1310	21	0.30	76/46	
1950	13	0.30	74/62	Travel rate 54 min/fsw. Air at 1700.
2310	9	0.26		RJ complains left ear swollen shut, uncomfortable with headphones.
2303	9	0.26	77/56	Doppler, 1 min each, no bubbles.
* * * * *	1975 March 26, Dive Day 15, Lab Day 14 * * * * *			
0247	5	0.24	76/46	Travel rate 0.20 in/min.
0318	5	0.24		RJ throbbing, pain in left ear, feels swollen shut; Auralgan given, ice bag sent in.
0320	5	0.24		MD (CH) notified of BJ's pain.
0342	4	0.24		RJ has some relief after applying antipyrine and benzocaine (1/2 stopper in ear), ice pack.
0416	4	0.24	77/45	
0656	2	0.22	76/43	Reveille. VS.
0820	0	0.21		Reach surface. Ceremony, photographs. NT has had high blood pressure last few days, swollen ear, tires easily.
1400	JB has athlete's foot; remembers taking off air mask, going on nitrox, thought he did well on performance but tests show otherwise. RJ left ear canal inflammed.			
* * * * *	1975 March 27, Lab Day 15 * * * * *			
	RJ thigh and groin pain, happened getting out of bed last night.			
* * * * *	1975 March 28, Lab Day 16 * * * * *			
	BJ and NT take exercise tests.			
* * * * *	1975 March 31, Lab Day 17 * * * * *			
	RJ leg pain diminished, ear pain gone. NT and JB feel well. All back to duty, NT to get hypertension test, RJ upper g.i. and gall bladder series.			

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
* * * * *	1976 June 8, Dive Day 1 * * * * *			
1003	0	0.21		Begin compression; subjects JC, MH, RO on mask.
1012	66			Reach bottom. Divers off mask. PCO_2=0.2 mmHg. No humidity readings on this dive.
1040	66		83/	No health problems. MH reports voice change.
1232	66		84/	MH ears OK; RO some fluid behind left ear.
1338	66		80/	Psychologist BW interviewing divers.
1655	66		81/	MH reports feeling sleepy; RO and JC feeling fine. Vital signs.
1732	66	0.31	79/	Subjects report general loss of tension after start of dive; RO report pain in left ear from prior equalization difficulties; MH reports he feels sleepy, has voice difficulties.
1804	66	0.31		Divers take ear drops, 5 min each ear.
1951	66	0.31	78/	MH takes Cepacol lozenges for sore throat.
2130	66	0.29	76/	MH receives Robitussin for throat, 2 tbsp/4 hr.
2245	66	0.29	80/	Lights out.
* * * * *	1976 June 9, Dive Day 2 * * * * *			
0605				Vital signs.
0900	66	0.29	77/	Divers get Domeboro, 5 min; PCO_2=0.2 mmHg.
1017	66	0.31	77/	MH notified of white cells in urine, no urinary complaints.
1300	66	0.31	77/	Psychologist BW interview, 5 min each; Rolaids sent in.
1646	66	0.30	78/	Vital signs.
1827	66	0.31	75/	MH pain in both ears, told to increase ear drops; Doppler.
2300	66	0.29	75/	JC, MH receive eardrops. PCO_2=1.1 mmHg. Lights out.
* * * * *	1976 June 10, Dive Day 3 * * * * *			
0624	66	0.30	72/	Vital signs. Eardrops for all. PCO_2=0.2 mmHg.
1200	66	0.29	78/	Divers get eardrops, nap.
1400	66	0.31	75/	Interviews. MH into body box for 6 min. PCO_2=2.2 mmHg.
1512	66	0.31	82/95	Refrigeration system repairs completed.
1617	66	0.31	82/	Divers take helium samples for mass spec.
1627	66			Mass spectrometer shows 9.2% He.
1651	66			Divers in outer lock, inner lock being flushed with air.
1738	66			Divers try passing through the diaphragm.
1844	66	0.31	79/	Eardrops.
1925	66	0.32	74/	Doppler, 29 min each, no bubbles reported.
2104	66	0.33	78/	JC reports feeling lightheaded and nauseated; mouth sore from parotid sampling.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
* * * * *	1976 June 11, Dive Day 4 * * * * *			
0022	66	0.30	77/	Lights out.
0625		0.29	74/	Vital signs.
0820	66	0.29	79/	MH requests more Robitussin cough syrup.
0915	66	0.30	79/	MH mouth sore from parotid study.
0923	66	0.30		Divers transfer to outer lock.
0925	66			Diaphragm in place on hatch, hatch closed.
0933	66	0.30	76/	Inner lock ascending to surface.
0940	66			Begin purge of inner lock with He.
0941	10			Inner lock N_2 88%
0951	10			Inner lock N_2 at top 6%, bottom 40%
0954	10			Inner lock N_2 at top 2%, bottom 37%
0958	10			Inner lock N_2 at top 0.6%, bottom 32%
1002	0			Inner lock N_2 at bottom 24%
1005	0			Inner lock N_2 at bottom 12.4%
1008	0			Inner lock N_2 at bottom 2.2%
1010	0			Inner lock N_2 at bottom 0.4%
1012	66	0.29	78/	Inner lock descending to 66 fsw depth on He. Divers receive eardrops (divers in outer lock).
1014	66	0.29	80/	Seal between locks broken.
1124	66	0.29	82/	JC in inner lock attempting to reseal diaphragm, Doppler 8 min, no bubbles. N_2 bottom inner lock 17%, He in outer 17%.
1132	66	0.28	82/	MH and RO report discomfort in outer lock; outer lock compressed to 67 fsw to maintain seal.
1202	66	0.30	81/	Project conference: MH will make transition as soon as plethysmograph is ready. RO will go on 90/10 Nitrox mixture by mask until transition. Decision based on unexpectedly high readings of He (17%) in outer lock and N_2 (17%) in inner lock.
1209	66	0.30	81/	RO on nitrox 90/10 mixture by mask, outer lock.
1219	66	0.30	81/	Doppler, JC 3 min, no bubbles reported.
1227	66			MH to inner lock.
1230	66	0.29	81/	MH in body box for 57 min; Doppler 8 min.
1243	66	0.28	81/	Doppler, JC 12 min, MH 9 min, MH 7 min at 1309.
1352	66	0.31	81/	EKG and Doppler, MH 9 min, no bubbles.
1410	66			RO to inner lock.
1420	66	0.30	80/	RO in body box for 69 min; Doppler, 6 min RO and MH, no bubbles reported.
1429	66		82/	Doppler all divers, no bubbles reported.
1452	66			Doppler MH and JC, no bubbles.
1457	66		80/	RO and JC have mild generalized itching, no rash.
1505	66		78/	Doppler RO 13 min, JC 5 min, MH, no bubbles.
1555	66	0.36	80/	MD (CH) orders all tests stopped, 5 min rest.
1631	66			JC reports mild itching, not localized.
1745	66	0.31		Doppler all; RO itch over lower body.
1809	66	0.32	82/	JC reports itching subsiding. Robitussin for MH.
1832	66	0.32		RO reports still itching.
2010			80/	Gauge malfunction, increased pressure to 80 fsw for 2 min to check.
2022				RO and MH report still have mild itching.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
* * * * *	1976 June 8, Dive Day 1 * * * * *			
1003	0	0.21		Begin compression; subjects JC, MH, RO on mask.
1012	66			Reach bottom. Divers off mask. PCO_2=0.2 mmHg. No humidity readings on this dive.
1040	66		83/	No health problems. MH reports voice change.
1232	66		84/	MH ears OK; RO some fluid behind left ear.
1338	66		80/	Psychologist BW interviewing divers.
1655	66		81/	MH reports feeling sleepy; RO and JC feeling fine. Vital signs.
1732	66	0.31	79/	Subjects report general loss of tension after start of dive; RO report pain in left ear from prior equalization difficulties; MH reports he feels sleepy, has voice difficulties.
1804	66	0.31		Divers take ear drops, 5 min each ear.
1951	66	0.31	78/	MH takes Cepacol lozenges for sore throat.
2130	66	0.29	76/	MH receives Robitussin for throat, 2 tbsp/4 hr.
2245	66	0.29	80/	Lights out.
* * * * *	1976 June 9, Dive Day 2 * * * * *			
0605				Vital signs.
0900	66	0.29	77/	Divers get Domeboro, 5 min; PCO_2=0.2 mmHg.
1017	66	0.31	77/	MH notified of white cells in urine, no urinary complaints.
1300	66	0.31	77/	Psychologist BW interview, 5 min each; Rolaids sent in.
1646	66	0.30	78/	Vital signs.
1827	66	0.31	75/	MH pain in both ears, told to increase ear drops; Doppler.
2300	66	0.29	75/	JC, MH receive eardrops. PCO_2=1.1 mmHg. Lights out.
* * * * *	1976 June 10, Dive Day 3 * * * * *			
0624	66	0.30	72/	Vital signs. Eardrops for all. PCO_2=0.2 mmHg.
1200	66	0.29	78/	Divers get eardrops, nap.
1400	66	0.31	75/	Interviews. MH into body box for 6 min. PCO_2=2.2 mmHg.
1512	66	0.31	82/95	Refrigeration system repairs completed.
1617	66	0.31	82/	Divers take helium samples for mass spec.
1627	66			Mass spectrometer shows 9.2% He.
1651	66			Divers in outer lock, inner lock being flushed with air.
1738	66			Divers try passing through the diaphragm.
1844	66	0.31	79/	Eardrops.
1925	66	0.32	74/	Doppler, 29 min each, no bubbles reported.
2104	66	0.33	78/	JC reports feeling lightheaded and nauseated; mouth sore from parotid sampling.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
* * * * *	1976 June 11, Dive Day 4 * * * * *			
0022	66	0.30	77/	Lights out.
0625		0.29	74/	Vital signs.
0820	66	0.29	79/	MH requests more Robitussin cough syrup.
0915	66	0.30	79/	MH mouth sore from parotid study.
0923	66	0.30		Divers transfer to outer lock.
0925	66			Diaphragm in place on hatch, hatch closed.
0933	66	0.30	76/	Inner lock ascending to surface.
0940	66			Begin purge of inner lock with He.
0941	10			Inner lock N_2 88%
0951	10			Inner lock N_2 at top 6%, bottom 40%
0954	10			Inner lock N_2 at top 2%, bottom 37%
0958	10			Inner lock N_2 at top 0.6%, bottom 32%
1002	0			Inner lock N_2 at bottom 24%
1005	0			Inner lock N_2 at bottom 12.4%
1008	0			Inner lock N_2 at bottom 2.2%
1010	0			Inner lock N_2 at bottom 0.4%
1012	66	0.29	78/	Inner lock descending to 66 fsw depth on He. Divers receive eardrops (divers in outer lock).
1014	66	0.29	80/	Seal between locks broken.
1124	66	0.29	82/	JC in inner lock attempting to reseal diaphragm, Doppler 8 min, no bubbles. N_2 bottom inner lock 17%, He in outer 17%.
1132	66	0.28	82/	MH and RO report discomfort in outer lock; outer lock compressed to 67 fsw to maintain seal.
1202	66	0.30	81/	Project conference: MH will make transition as soon as plethysmograph is ready. RO will go on 90/10 Nitrox mixture by mask until transition. Decision based on unexpectedly high readings of He (17%) in outer lock and N_2 (17%) in inner lock.
1209	66	0.30	81/	RO on nitrox 90/10 mixture by mask, outer lock.
1219	66	0.30	81/	Doppler, JC 3 min, no bubbles reported.
1227	66			MH to inner lock.
1230	66	0.29	81/	MH in body box for 57 min; Doppler 8 min.
1243	66	0.28	81/	Doppler, JC 12 min, MH 9 min, MH 7 min at 1309.
1352	66	0.31	81/	EKG and Doppler, MH 9 min, no bubbles.
1410	66			RO to inner lock.
1420	66	0.30	80/	RO in body box for 69 min; Doppler, 6 min RO and MH, no bubbles reported.
1429	66		82/	Doppler all divers, no bubbles reported.
1452	66			Doppler MH and JC, no bubbles.
1457	66		80/	RO and JC have mild generalized itching, no rash.
1505	66		78/	Doppler RO 13 min, JC 5 min, MH, no bubbles.
1555	66	0.36	80/	MD (CH) orders all tests stopped, 5 min rest.
1631	66			JC reports mild itching, not localized.
1745	66	0.31		Doppler all; RO itch over lower body.
1809	66	0.32	82/	JC reports itching subsiding. Robitussin for MH.
1832	66	0.32		RO reports still itching.
2010			80/	Gauge malfunction, increased pressure to 80 fsw for 2 min to check.
2022				RO and MH report still have mild itching.

Time of day	Depth fsw	P_{O_2} atm	t_F, R.H.%	Remarks
* * * * *	1976 June 23, Dive Day 1 * * * * *			
1015	0	0.21		Begin 9 min compression to 99 feet; 3 divers TG, RL, RE; divers on mask. No narcosis reported.
1103	99	0.31	78/	TG reports cramp in groin, transient.
1116	99	0.31		RL has pain in right arm, popping, at old break (2 min); has trouble concentrating on task.
1121	99	0.31		TG reports groin still cramped.
1204	99	0.31	76/	RL reports pain in right arm stopped.
1642	99	0.31		Vital signs.
1800	99	0.31	76/	RE takes 1 pinch of Skoal snuff. Eardrops for all. PCO_2=0.6 mmHg. TG takes penicillin tablet.
1926	99	0.31	75/	Doppler/EKG, 26 min, no bubbles reported.
2155	99	0.31	74/	Taps.
* * * * *	1976 June 24, Dive Day 2 * * * * *			
0630	99			Vital signs.
0800	99	0.31	77/	TG takes medicine, changes foot dressing.
0907	99	0.32	76/	All divers receive eardrops. PCO_2=1.0 mmHg.
1041	99	0.30	77/	RL complains of being cold.
1210	99	0.30	78/	TG takes medicine. All divers receive eardrops. PCO_2=1.0 mmHg.
1334	99	0.30	78/	TG has headache, lasts about 1/2 hour.
1500	99	0.30	79/	Psych; vital signs at 1600.
1720	99			TG feels stiffness in right thumb joint, present all day; RL has pain in feet, possibly fungus, feels tired. Eardrops for all at 1800.
1934	99	0.30	84/78	RL has athlete's foot; MD gives treatment.
2223	99	0.30	78/	RG takes penicillin. Taps 2224.
2352	99	0.30	76/	RL reports severe headache over left eye, receives aspirin and ice pack.
* * * * *	1976 June 25, Dive Day 3 * * * * *			
0635	99	0.31	75/	Vital signs.
0831	99	0.31	75/	Eardrop time. TG takes penicillin.
1035	99	0.31	78/	TG reports foot doing well (rugby injury), no drainage; swelling down.
1100	99	0.29	78/74	Psychologist BW interviews divers for 8 min.
1245	99	0.31	80/	All divers receive eardrops. PCO_2=0.6 mmHg.
1300	99	0.31	81/	TG takes penicillin.
1858	99	0.31	81/69	Doppler, no bubbles. Eardrops; vital signs 1818.
2148	99	0.31	79/	TG takes penicillin, changes dressing. Taps.
* * * * *	1976 June 26, Dive Day 4 * * * * *			
0600	99	0.31	75/	Vital signs.
0900	99	0.30	77/79	
0909	99	0.30	74/	TG and RE put on suits, go on air masks in inner lock; suits plugged in at 0917.
0920	99			TG and RE report suit and air hookup completed.
0940	99			Purging inner lock with helium; RL in outer lock.
0958	99			TG headache in top of head; feels hot.
1007	99			RL reports outer lock smells funny, medicinal.
1011	99		76/	TG headache clearing up, not as hot.
1027	99			Suits were leaking air into inner chamber.
1040	99			RL has headache.
1113	99		78/51	RL headache gone, smell also gone; O.L. 76F.
1121	99	0.29		TG removes hood and suit, reports feeling fine.
1202	99	0.28	78/	RL puts on suit, on air in outer lock.
1209	99			TG reports stiffness in joints of fingers, hard to bend fingers.
1217	99			TG reports itching; Doppler 2 min, no bubbles.
1221	99			TG reports itching on arms and legs; slight rash on arms; takes off his shirt.
1225	99			Ears popping, like during depth changes.
1227	99			RE comes off mask, takes off his suit.
1230	99			RE has hood off, on helium.
1239	99			RE reports itching; RL reports headache.
1245	99			TG takes his scheduled penicillin; has headache, itching on arms, skin a little red, rash on legs; RE reports some redness on back.
1252	99	0.29		TG has "hell of a headache;" fingers clearing up.
1304	99		76/	RL vents suit; RL feet going to sleep.
1315	99			RL reports feeling pressure between eyebrows.
1326	99			TG reports itching, rash, and headache clearing up; RL reports starting to itch.
1401	99	0.31	77/	RL off mask, hood removed, moves to inner lock.
1408	99			RL feels dizzy after removing suit, "room moved;" no hearing problem, no itching, no nausea.
1431	99			RE in suit except hood, filling suit with N_2, feels bad, hot. Doppler, 10 min TG, no bubbles.
1438	99			RE reports itch diminished.
1440	99			RL starting to itch, rash on arms.
1443	99			RE removes suit, has blotchy red welts on shoulder, pencil point size like mosquito bites; taking pictures.
1450	99			RE reports leg itching, welts receding.
1452	99			RL has raised bumps on back.
1456	99			RE itching gone; TG itching on back of leg.
1500	99	0.30	80/	RE reports both legs itching.
1556	99	0.31		RL dressing in suit, with gloves. Suit purged with nitrogen for 30 seconds. Doppler, 13 min RL and RE, no bubbles.
1600	99			RL reports arms and legs feeling better in nitrogen-filled suit, itching almost gone.
1602	99		77/	RL reports sore skin from scratching. Nitrogen vent for 30 seconds. RL feeling much better.
1614	99			Doppler 4 min RL, no bubbles reported.
1621	99			RL reports return of bad itch, arms and legs, no rash; RE rash still present; TG itch improving.

Time of day	Depth fsw	PO_2 atm	Temp °F, R.H.%	Remarks
1645	99	0.30		Re crepitation: (Score: 1=barely audible; 5=loud) TG +2 both thumbs, +3 middle fingers; RL +1 both index fingers; RE +1 several joints.
1651	99			Itching: TG none; RL continuing; RE slight. Vital signs and Doppler.
1742	99	0.30	83/68	Doppler 3 min TG, no bubbles, takes penicillin.
1817	99			All subjects report itching has ceased.
2004	99			RE headache at back of head for 2 hr, since coming off respiratory gear; TG crept'n: thumbs +3 Rt, +4 left, +1 or +2 other fingers; wrists +2; Left shoulder +2, Rt +3; knees +4. RL sore thighs, rt lower back when he moves.
2051	99	0.30	85/	CH & DK medical summary: RL no symptoms. TG pain in both knees at top above cap, present at rest increases while standing or walking, not sensitive to touch, mild to moderate intensity; no other joint signs. RE complains of mild, dull pain in both knees for 90 min duration, pain, absent at rest but present when standing or bending; headache gone.
2059	99	0.30	85/	PCO_2 has increased to 4 mmHg.
2140	99	0.30		DK medical summary: RE no knee pain. TG pain in right knee decreased, waxes and wanes; pain in left knee steady, worse when bending.
2232	99	0.30		Doppler RE and RL, nothing.
2332	99	0.30	84/	Medical summary: TG pain in left knee worse, right knee same, MD plans to treat with hyperoxic mix.
2347	99	0.30	85/	TG on 51-49% He-O_2 for 20 min; TG reports discomfort in right ankle at start of treatment.
* * * * *	1976 June 27, Dive Day 5 * * * * *			
0000	99	0.30		TG reports right knee and ankle feeling better, left knee feels the same.
0007	99	0.30	86/	TG off treatment mix.
0013	99	0.30	86/	TG on mix for 19 min; leg cramps, right worse, left painful when moved.
0032	99	0.30		TG off mask.
0037	99	0.30		All on mask to compress to 109 fsw at 1 fsw/min. TG on O_2 for 23 min, others off at 109.
0041	104			TG reports left knee better; right knee worse.
0101	109	0.31	86/	TG on O_2 for 20 min; pressurize to 129 fsw.
0108	123			TG reports pain in knee constant.
0112	129			TG knee pain worsening, very painful to touch.
0120	129			Slight improvement left knee, little pain in rt.
0128	129			TG on mask for 20 min.
0142	129			RL and RE complain of itching.
0153	129			TG on mask.
0159	129			RL and RE itching has terminated. Vital signs.
0213	129			TG off mask; pain almost gone in right knee.
0219	129			RL reports itching, TG reports red, bumpy rash on arm and legs. Doppler, 3 min RE and RL, 2 min TG, no bubbles reported.
0252	129	0.30	82/	RL reports itching subsided.
0318	129			TG reports no pain in left knee, slight pain in right knee. Lights out.
0422	129	0.29	80/	RE itching on neck and legs.
1000	129	0.24	76/	Wake up, all fine but tired. RL still has rash, vital signs.
1041	129	0.30	83/	RL reports rash is gone.
1157	129	0.31		TG takes penicillin.
1239	129			TG changes dressing on foot. Eardrops 1300, all.
1510	129	0.31	83/	Doppler 7 min RE and RL, 2 min TG, no bubbles.
1612	129	0.30	83/77	Vital signs.
1810	129	0.30	83/	Doppler 7 min each, no bubbles.
2307	129	0.30		TG changed dressing, took penicillin. Taps.
* * * * *	1976 June 27, Dive Day 6 * * * * *			
0501	129	0.30		Wake up, vital signs.
0600	129	0.30	82/	Begin decompression.
0700	125	0.32	83/	Divers receive eardrops.
1100		0.32	83/	Doppler, 13 min each, no bubbles.
1231	103	0.37	84/	TG takes penicillin. All take eardrops.
1353	97	0.33	86/69	Reach 97 feet, hold for 2 hours.
1638	94			Vital signs. Eardrops at 1750.
2053	77	0.30		Doppler, 13 min each, no bubbles.
2057	77	0.33		RL has mild red rash on legs skin tender to touch from scratching.
2215	72	0.33	86/	Taps.
2354	65	0.33		Hold at 65 fsw till 0600 while divers sleep.
* * * * *	1976 June 28, Dive Day 7 * * * * *			
0600	65	0.33	79/	Resume decompression.
0704	60	0.32	79/	Vital signs.
0826	55	0.32		TG changes dressing on foot and takes his pill.
0909	52	0.33		Divers take ear drops.
0930	51			Ascent rate now 3 fsw/hr.
1034	44	0.33		Doppler, 4 min TG, 3 min RE, 2 min RL, no bubbles.
1223	42	0.32		TG takes pill.
1400	37	0.32	78/70	Hold for 2 hours.
1631	35	0.33		Vital signs.
2236	17	0.33	81/	TG takes pill. Taps 2240.
2400	13	0.32		Hold 6 hr.
* * * * *	1976 June 29, Dive Day 8 * * * * *			
0621	10	0.33	83/	Vital signs.
0742	8	0.25	81/84	TG takes pill.
1020	0	0.21	72/	Reach surface.

APPENDIX B
MEDICAL DIVING LOCKER
As of the time of Nisat I

1. Diagnostic equipment

All routine diagnostic equipment including: blood pressure cuff, stethoscope, otoscope, ophthalmoscope, reflex hammer, tuning fork, tongue blades, sensory testing equipment

2. Supplies

Laryngoscopes
Endotracheal tubes
Foley catheter and drainage bag set up
C.V.P. needles, and manometers
3-way stop cocks
Cutdown tray
Syringes (from 5 to 50 cc) and needles
Thoracotomy tray
Tracheostomy tray
Intracath needles, 16G to 20G
Ambu bag and mask
Foot operated aspirator
Tape, suture material, etc
Naso-gastric tubes

3. Drugs and fluids

LMW dextran
Lactated Ringer's solution
5% dextrose in water
Decadron
Epinephrine
Valium
Mannitol
Sodium bicarbonate
Aramine
Isuprel
Atropine
Aminophyllin
Hydrocortisone
Lasix
Lanoxin
Compazine

Common drugs such as ASA (aspirin), Sudafed, Auralgan, Cortisporin Otic.

4. In-chamber medicine

No medicines were allowed in the chamber unless approved by the Medical Officer of the Watch, and then for a single dose only, except two bottles of eardrops (Domeboro Otic) for each diver, clearly labelled "left" and "right" and with his name.

5. In-Chamber Medical Kit

Stethoscope
Oto/ophthalmoscope (with operating head)
Sphygmomanometer (vented)
Disposable needles
Tuning Fork (254 cps)
Plastic oral airway
Padded tongue blade
Reflex hammer
Cotton ball
Ambu bag kit

Unclassified

SECURITY CLASSIFICATION OF THIS PAGE *(When Data Entered)*

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER NSMRL Report 985	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER
4. TITLE *(and Subtitle)* SHAD-NISAT: A Composite Study of Shallow Saturation Diving incorporating Long Duration Air Saturation with Excursions, Deep Nitrox Saturation and Switch from Nitrogen to Helium		5. TYPE OF REPORT & PERIOD COVERED Report on dive Projects 6. PERFORMING ORG. REPORT NUMBER NSMRL Report 985
7. AUTHOR(s) R. W. Hamilton, G. M. Adams, C. A. Harvey and D. R. Knight (editors)		8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS Naval Submarine Medical Research Laboratory Box 900 Naval Submarine Base New London Groton, Connecticut 06349		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS See reverse side
11. CONTROLLING OFFICE NAME AND ADDRESS same as 9		12. REPORT DATE August 1982 13. NUMBER OF PAGES 180
14. MONITORING AGENCY NAME & ADDRESS*(if different from Controlling Office)* Naval Medical Research and Development Command National Naval Medical Center Bethesda, Maryland 20814		15. SECURITY CLASS. *(of this report)* Unclassified 15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT *(of this Report)*

Approved for public release; distribution unlimited

17. DISTRIBUTION STATEMENT *(of the abstract entered in Block 20, if different from Report)*

18. SUPPLEMENTARY NOTES

19. KEY WORDS *(Continue on reverse side if necessary and identify by block number)*

Adaptation; bradycardia; counterdiffusion; breathing mixtures; decompression; decompression sickness; ECG; EEG; erythrocytes; excursion diving; helium; hyperoxia; itching; narcosis; nausea; nitrogen; oxygen toxicity; performance; saturation; selection; tables

20. ABSTRACT *(Continue on reverse side if necessary and identify by block number)*

Seven dry chamber laboratory exposures at NSMRL explored the feasibility, physiological effects, safe limits, and operational procedures applicable to air and nitrogen-oxygen saturation diving. Objectives included establishing Navy capability in nitrogen-based saturation diving and improving certain submarine rescue procedures. The four SHAD dives looked at air for breathing in saturation at 50 and 60 fsw. Excur-

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE
S/N 0102-014-6601 |

Unclassified

SECURITY CLASSIFICATION OF THIS PAGE *(When Data Entered)*

continuation of item 20.

sions from these depths demonstrated possible work procedures, covering depths ranging between 5 and 250 fsw and for times as long as 8 hours. The exposures were well tolerated but disclosed problems with oxygen toxicity in daily 8-hour excursions to 100 fsw and an increased sensitivity to extra oxygen breathing in several of the divers. The long air exposures caused red blood cell losses; recovery began a few days after return to normal pressure. The divers were also deconditioned, presumably because of the confinement and several weeks of inactivity. Decompressions from descending excursions were free of bends but some ascending excursions caused itching and ultrasonically-detectable bubbles. Two of three divers became nauseated 3 hours after beginning a saturation exposure in an atmosphere containing 0.22 atm oxygen, balance nitrogen, at 7 atm abs; the sick divers felt better after PO_2 was raised to 0.3 atm. All 3 of these divers were "drunk" for several days as a result of nitrogen narcosis, but recovered many aspects of normal performance after 5 - 6 days. In two experiments divers saturated with nitrox (0.3 atm PO_2) at 66 and 99 fse were switched to a helium mixture. Itching followed within 3 hours, and was quite intense for the 99 fsw crew; one of these had to be treated by recompression for "counterdiffusion sickness" manifested as knee pain. Hyperbaric bradycardia was observed in most of the divers, intensifying on the deeper excursions.

- - - - - - - -

Item 10 -- Work Units -- M4306.02-3114.BEK9.02; M4306.01-8013; MPN10.003-7060; MR041.01-5057; MF51.524.014-9018; M4306.01-2001BFM9, and M4306.02-2110BAK9